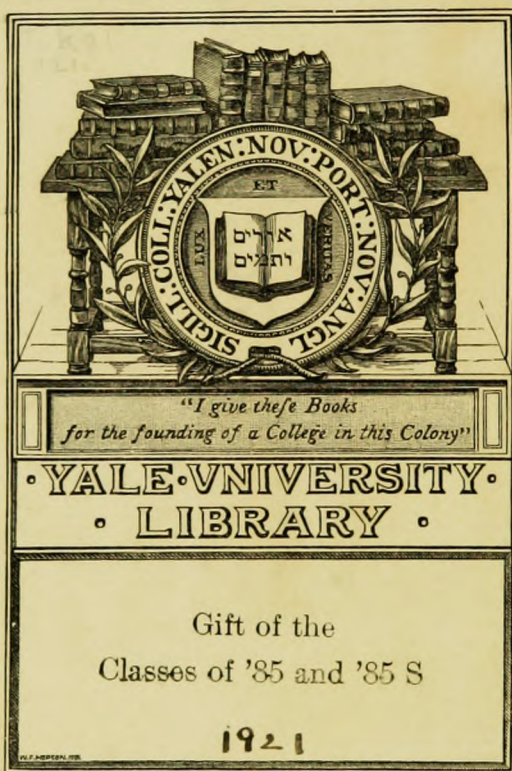


YALE UNIVERSITY LIBRARY



3 9002 06606 6250





TRANSFERRED TO  
YALE MEDICAL LIBRARY















# NERVOUS AND MENTAL DISEASES

BY

ARCHIBALD CHURCH, M.D.

Professor of Nervous and Mental Diseases in the Northwestern University Medical School,  
Chicago; Formerly Professor of Neurology in the Chicago Polyclinic;  
Neurologist to St. Luke's, Wesley, Mercy, and  
Michael Reese Hospitals, etc.

AND

FREDERICK PETERSON, M.D.

Ex-President of the New York State Commission in Lunacy; Formerly Professor of Psychiatry,  
Columbia University; Consulting Alienist, Bellevue Hospital; Manager of the  
Craig Colony for Epileptics at Sonyea, New York; Ex-President  
of the New York Neurological Society.

---

WITH 350 ILLUSTRATIONS

---

*NINTH EDITION, THOROUGHLY REVISED*

PHILADELPHIA AND LONDON  
W. B. SAUNDERS COMPANY

1921

COPYRIGHT, 1899, BY W. B. SAUNDERS. REPRINTED OCTOBER, 1899. REVISED, REPRINTED, AND  
RECOPYRIGHTED JULY, 1901. REPRINTED APRIL, 1902. REVISED, REPRINTED, AND RECOPY-  
RIGHTED AUGUST, 1903. REPRINTED MAY, 1904. REVISED, REPRINTED, AND  
RECOPYRIGHTED AUGUST, 1905. REPRINTED JUNE, 1906, AND OCTOBER,  
1907. REVISED, REPRINTED, AND RECOPYRIGHTED AUGUST, 1908. RE-  
PRINTED MAY, 1910, AND OCTOBER, 1910. REVISED, REPRINTED,  
AND RECOPYRIGHTED, OCTOBER, 1911. REVISED, RE-  
PRINTED, AND RECOPYRIGHTED SEPTEMBER, 1914.  
REPRINTED DECEMBER, 1915. REVISED,  
REPRINTED, AND RECOPYRIGHTED,  
OCTOBER, 1919.

---

COPYRIGHT, 1919, BY W. B. SAUNDERS COMPANY

---

Reprinted January, 1921

RC341  
921C

A.B.

---

PRINTED IN AMERICA

---

PRESS OF  
W. B. SAUNDERS COMPANY  
PHILADELPHIA



## PREFACE TO THE NINTH EDITION.

---

IN the preparation of this edition no radical changes have been made. Guided by the kindly advice of a large number of medical teachers, the same general presentation of neurological subjects has been retained. Some radical views which have attained considerable popularity have not been incorporated, more time and experience being thought requisite for their unquestioned establishment.

The subjects of General Paresis and Traumatic Insanity have been rewritten and many interpolations and minor corrections made. The needs of the student and the wants of the practitioner have had first consideration throughout.

The continued demand for the book is a great gratification to the authors.





## PREFACE.

---

THIS book has been written for medical students and general practitioners. It makes no claim to be other than a carefully prepared text-book. The literature of neurology and psychiatry has been sifted by the authors, and such digest revised in the light of their own experience in practice and in teaching. They have attempted to present their facts clearly, directly, and with brevity, despite the difficulty of condensing two great subjects within the limits of a single volume.

This is not the joint work of two writers, but each author—Dr. Church in Neurology, and Dr. Peterson in Psychiatry—has contributed to the making of a single volume what might have made a separate monograph; each is, therefore, solely responsible for the work in his own department. In placing the correlated sciences neurology and psychiatry under the same cover, the reader's convenience was considered.

An unusual number of illustrations for each department (from the authors' own material, except when otherwise indicated) has been allowed by a generous publisher.





# CONTENTS.

## NERVOUS DISEASES.

### PART I.

	Page
EXAMINATION OF PATIENTS	17
Chapter I. The Anamnesis	17
Chapter II. The General Physical Examination	21
Chapter III. The Muscular System	26
Errors of Motility	28
The Reflexes	32
Chapter IV. Trophic Conditions	39
Chapter V. Electrical Conditions	41
Chapter VI. Sensory Conditions	49
The Tactile Sense	50
The Pain Sense	54
The Pressure Sense	54
The Thermal Sense	54
The Muscular Sense	54
Pain	57
Chapter VII. The Special Senses	62
Sight	62
Hearing	65
Smell	66
Taste	66
Chapter VIII. Speech	67

### PART II.

DISEASES OF THE CEREBRAL MENINGES AND CRANIAL NERVES	72
Chapter I. Cerebral Meninges	72
Pachymeningitis	74
Pia Hemorrhage	76
Chapter II. Inflammation of the Pia Mater	78
Acute Leptomeningitis	78
Varieties of Leptomeningitis	80
Diphtheritic Meningitis	80
Tubercular Meningitis	80
Serous Meningitis	80
Chronic Leptomeningitis	87
Chapter III. Diseases of the First and Second Cranial Nerves	88
Chapter IV. Diseases of the Ocular Nerves	107
Chapter V. Diseases of the Trifacial Nerve	110
Chapter VI. Diseases of the Facial Nerve	121
Chapter VII. Diseases of the Eighth Cranial Nerve	130
Chapter VIII. Diseases of the Glossopharyngeal, Vagus, and Accessory Nerves	138
Chapter IX. Diseases of the Spinal Portion of the Accessory Nerve	149
Chapter X. Diseases of the Hypoglossal Nerve	152
Chapter XI. Multiple Paralysis of Cranial Nerves	154

## PART III.

	PAGE
DISEASES OF THE BRAIN PROPER.....	165
Chapter I. The Cerebral Cortex—Localization.....	165
Chapter II. Speech and the Cortex—Aphasia.....	174
Chapter III. The Cerebral White Matter, Basal Ganglia, and Cerebellum.....	184
Chapter IV. Further Localizing Considerations.....	192
Chapter V. Arterial Brain Diseases.....	197
Cerebral Anemia and Hypertension.....	199
Cerebral Arteritis.....	202
Chapter VI. Cerebral Hemorrhage and the Hemiplegic State.....	207
Chapter VII. Cerebral Softening.....	222
Chapter VIII. Diseases of the Cerebral Vessels and Sinuses.....	251
Chapter IX. Cerebritis, Encephalitis, and Abscess of the Brain.....	258
Chapter X. The Cerebral Pulses of Children.....	263
Chapter XI. Tumors of the Brain.....	266
Chapter XII. Hydrocephalus.....	271

## PART IV.

DISEASES OF THE SPINAL MENINGES AND SPINAL NERVES.....	276
Chapter I. Spinal Meningitis and Spinal Meningeal Hemorrhage.....	276
Chapter II. Injuries and Diseases of Spinal Nerves.....	284
Chapter III. Lesions of Spinal Spinal Nerves.....	295
The Cervical and Brachial Plexuses.....	295
Combined Palsies of the Nerves of the Arm.....	302
Nerves of the Trunk.....	306
Nerves of the Lower Extremity.....	307
Chapter IV. Multiple Neuritis.....	316
Chapter V. Horner's Zoster.....	320

## PART V.

DISEASES OF THE CON. PHASE.....	340
Chapter I. Localization.....	340
Chapter II. Indiscriminate Cord Lesions.....	362
Traumatic Lesions of the Cord Substance.....	362
Hemorrhage into the Cord.....	362
Thrombotic Softening of the Cord.....	364
Myelitis.....	365
The Paraplegic State.....	373
Lindsay's Paralysis.....	376
Caisson Disease, or Drivers' Palsy.....	380
Tumors of the Spinal Cord and Its Envelopes.....	382
Spina Bifida.....	386
Chapter III. Lesions and Diseases of the Spinal Gray Matter.....	388
Acute Anterior Poliomyelitis.....	388
Syringomyelia.....	390
Progressive Muscular Atrophy Showing Lesions of the Spinal Gray Matter.....	407
Progressive Muscular Atrophy Without Striking Conclusions.....	415
Arthritic Muscular Atrophy.....	426
Chapter IV. Lesions Affecting the White Matter of the Cord.....	428
Tabes Dorsalis.....	429
Combined Sclerosis of the Spinal Cord.....	464

## Chapter IV. (Continued).

Combined Conditions in Ataxia and Cerebriasis.....	468
Family Ataxia.....	470
Hereditary Spastic Paraplegia.....	476
Sclerosis of the Cord Due to Vegetable Intoxicants.....	477

## PART VI.

DISEASES OF GENERAL NERVOUS SYSTEM WITH KNOWN ANATOMICAL BASIS.....	481
Chapter I. Multiple Cerebrospinal Sclerosis.....	481
Chapter II. Syphilis of the Nervous System.....	490
Cerebral Syphilis.....	492
Spinal Syphilis.....	498
The So-called Parasyphilitic Diseases.....	503

## PART VII.

THE NEUROSES.....	505
Chapter I. Glandular Neuroses.....	506
Acromegalia.....	507
Adiposity and Genital Dystrophy.....	511
Adiposis Dolorosa.....	515
The Pituitary Syndrome.....	517
Trophoneuroses Related to the Thyroid and Parathyroid Glands.....	525
Myxedema.....	528
Exophthalmic Goiter.....	528
Scleroderma.....	539
Tetany.....	542
Chapter II. Vascular Neuroses.....	547
Raynaud's Disease.....	547
Anosmia.....	550
Intermittent Limping.....	550
Angioneurotic Edema.....	552
Chapter III. Hypertrophic Neuroses.....	555
Hypertrophic Cerebra.....	555
Hypertrophic Osteoarthralgia.....	556
Localized Hypertrophies.....	557
Chronic Hereditary Tropho-edema.....	560
Chapter IV. Infection Neuroses.....	563
Tetanus.....	568
Hydrophobia.....	569
Chorea.....	568
Chapter V. Motor Neuroses.....	578
Bartington's Disease.....	578
Myoclonia.....	579
Dakin's Disease.....	581
Parkinson's Disease, Paralysis Agitans.....	582
Thomson's Disease (Myotonia).....	588
Family Periodic Paralysis.....	591
Family Tremor.....	593
Chapter VI. Fungus Neuroses.....	593
Werners' Chorea.....	593
Other Fungus Neuroses.....	599
Chapter VII. Neurosthenia.....	600
Chapter VIII. Psychasthenia.....	620
Chapter IX. Motor Tic.....	624



Chapter X. Hysteria.....	618
Stigmata of Hysteria.....	621
Accidents of Hysteria.....	629
Chapter XI. Epilepsy.....	637
Chapter XII. Migraine.....	661
Chapter XIII. Neuroses Following Traumatism.....	666

## PART VIII.

Symptomatic Disorders.....	668
Chapter I. Neuralgia.....	668
Chapter II. Disorders of Sleep.....	674
Wakeful Disorders of Sleep.....	676
Somnolent Disorders of Sleep.....	680
Sleep Disorders.....	681
Hypnotism.....	682

## MENTAL DISEASES.

Chapter I. Definition and Classification of Insanity.....	687
Chapter II. General Etiology of Insanity.....	697
Sex; Age; Heredity.....	697
Strain.....	721
Chapter III. General Symptomatology of Insanity.....	737
Disorders of Sensation.....	738
Disorders of Ideas.....	744
Affective Disorders.....	746
Disorders of the Idea-associations.....	749
Disorders of Actions.....	758
Accompanying Physical Disorders.....	760
Chapter IV. Methods of Examination.....	765
Family History and History of the Patient.....	767
Observation of the Patient.....	768
Examination of the Patient's Physical and Mental Condition.....	770
Chapter V. General Treatment of Insanity.....	776
Treatment of Acute Cases.....	781
Chapter VI. Manic-depressive Insanity.....	790
The Manic Phase.....	793
The Depressive Phase.....	796
The Circular or Alternating Type.....	803
Imitation Melancholia.....	808
Chapter VII. Dementia Parva.....	811
The Hebephrenic Form.....	816
The Catatonic Form.....	817
The Paranoïd Form.....	818
Chapter VIII. Senile Dementia and Other Senile Psychoses.....	819
Senile Dementia.....	820
Other Senile Psychoses.....	822
Chapter IX. Paralytic Dementia.....	822
Chapter X. Pyramia.....	836
Chapter XI. The Neuropsychoses.....	840
Hysterical Insanity; Epileptic Insanity.....	849
Chapter XII. Intoxication.....	868
INDEX.....	929

# NERVOUS DISEASES.

BY

ARCHIBALD CHURCH, M. D.





# NERVOUS DISEASES.

## PART I.

### EXAMINATION OF PATIENTS.

#### CHAPTER I.

#### THE ANAMNESIS.

DIAGNOSTIC investigation in neurological work is a matter of painstaking care and thoroughness. A correct opinion depends upon it. It is the first step toward treatment, the legitimate end of medicine. The physician is dealing with morbid conditions, revealing themselves for the most part by functional errors. The large subjective side of the problem, with the unreliability of the patient's response, adds to the difficulty and calls for keenness of judgment at every step. The use of several tests for determining a given state is of much importance, especially in conditions that are not objectively evident. By comparison of the results thus obtained, and sometimes by striking an average of such results, exactness may be approximately reached, while dependence upon any one of them might be misleading.

On the other hand, by the use of instruments of precision, controlled by anatomical and physiological rules, a definiteness can often be reached in nervous diseases not elsewhere possible. Localization in brain and cord lesions is sometimes exact to a degree, and prognosis is often absolutely clear. But in many instances of the so-called functional diseases, careful study, the shrewdest judgment, and a wide experience enable one only approximately to appreciate the situation. The entire data can be secured only by a systematic and frequently prolonged or often-repeated examination, and it is of the first importance that the medical man should maintain an entirely judicial and non-committal mental attitude toward his patient and the diagnosis until he has every available fact at his disposal. Snap-shot diagnoses may be gratifying to all concerned, if correct, but they are very likely to be wrong and prejudicial to a proper subsequent estimation of the case and are never necessary.

A systematic examination can not be made without a prearranged formula. The nearer this corresponds to the development of the case, the less likely are important matters to be overlooked. It therefore should be chronological. Most patients insist on telling their own stories in their own way. It is sometimes well, especially in private practice, to allow them to do so, and when they finish, to begin

properly. In nervous diseases the family history is often of paramount importance. Taken with the appearance of the patient, it gives valuable indications as to the constitutional make-up of the individual. The family history, then, is to be investigated first, after noting the *name, age, sex, nationality, occupation, and social state* of the patient.

**Neurotic Heredity.**—In seeking information regarding the antecedents of a patient, much tact must sometimes be employed. Patients are loath to detail matters of this character, not always from a wish to conceal them, but from disinclination to admit even to themselves any serious shortcoming or morbidity. To the half that is directly learned an equal amount may sometimes be reasonably added. Much can be learned by interrogating other members of the family, especially if related by marriage, the family physician, and old acquaintances, but the confidence and rights of the patient must not be forgotten. Nor is it sufficient to seek for instances of the identical disease in the family history of the case. The significant factor is a *neuropathic liability*, and this is indicated with more or less force by the appearance of nervous and mental diseases of any sort, of slight or serious degree, in the ascendants. For instance, a hysterical mother has an epileptic child and an idiotic grandchild; or highly wrought nervous organizations in mother and father eventuate in neurasthenic and unstable children. *Idiosyncrasy* in mental and nervous diseases is quite common. *Insanity* in the same or neighboring generations may alternate with neuroses or mild psychoses, and any combination may exist. *Consanguinity* on the part of parents, in addition to furnishing a tendency to infecundity, is likely to bring together the subjects of similar neurotic taints, which are thereby reinforced in the offspring. In itself, however, consanguinity has probably been overestimated as a causal factor in nervous and mental diseases. The association of crime, perversity, genius, neuroses, and psychoses in related individuals may be encountered. Certain nervous diseases are of direct hereditary character, being transmitted from generation to generation or appearing in a group of cases in a given generation. It is only needful to mention Friedreich's ataxia and Huntington's chorea, but tabes dorsalis, paralysis agitans, multiple sclerosis, muscular atrophies, and practically every organic and functional nervous disease occasionally presents family groups of this nature.<sup>1</sup> Many of the familial diseases which have been described as essentially distinct are found to insensibly merge through the observation of intermediate cases; and Baerlin<sup>2</sup> contends that all typical diseases of a familial character drift into each other through such connecting links. A family constitutional defect is the only essential feature.

*Debilitating diseases*, like tuberculosis, rheumatism, and gout, are significant. Indeed, Charcot was inclined to consider arthritis as a congener of nervous diseases. *Tubercled syphilis* is on an entirely different footing. Not only is it capable of producing embryological

<sup>1</sup> Ch. Féré, "La Famille Neuropathique," Paris, 1904.

<sup>2</sup> "Deut. Zeit. f. Nervenk.," Oct., 1901.



defects, but its blight may make itself evident on the part of the nervous apparatus during the period of growth or in adult life, modifying cerebral or spinal functions and at times leading to histological changes in the central and peripheral parts, which may vary in degree up to destructive lesions. *Diabetes* and *Brigh's disease* are very common in neurotic families.

**Personal History.**—The investigator should go most carefully into the medical life-history of his patient. While doing so, indeed, whenever opportunity offers, the *countenance, attitude, manner, gait, posture, complexion, expression, gestures, and intelligence* of the person should be keenly watched. This observation becomes in time a trained, almost automatic, faculty, so that minute details subconsciously apprehended at the time can be readily recalled.

*In infancy*, was there any birth difficulty, possible brain or spinal injury from protracted labor, precipitate labor, or instrumentation; lack of vigor, suspicion of syphilis, or convulsions? During childhood, did the patient present any nervous phenomena, such as marked delirium or spasms under febrile conditions or from irritation of the gums and intestinal tract? Was there emesis, chorea, somnambulism, or night-terrors? Was he precocious or otherwise, docile or obstinate, cheerful or morose, forward or retiring? At *pubescence*, were there mental changes of unusual character, moodiness, expansiveness, cruelty? Was the establishment of menstruation attended by pain or hysterical manifestations? Was masturbation indulged in or suspected? During *adulthood*, what was the career, relation to the opposite sex, success in school and business, and what has been the course of events through *adult life*?

The *past illnesses* of the patient should then receive attention in the same systematic manner. The fevers and febricula of childhood, the exanthemata and infections. The diseases of the nasopharynx, stomach, intestines, and rectum, of the lungs and heart, of the skin, the special disorders of chest, abdomen, pelvis, and genito-urinary apparatus must not escape attention. Especial inquiry should be made for rheumatism, gonorrhea, grip, and malaria. The *present history* of the patient and the possibility of specific infection should in every case be carefully inquired into. This is a rule that has no exceptions. The investigation must be modified and guarded according to individual circumstances, but nothing should deter the physician from making sure that a factor of this sort is not overlooked. If injuries have been received, what were their character and consequences? Regarding *habits*, it is to be borne in mind that neurotic people are especially liable to carry everything to extremes and are prone to become the subject subjects of some perverted practice or stimulant addiction. Masturbation and venery take firm hold on them. Tobacco, alcohol, morphia, cocaine, chloral, and even tea and coffee master them completely. These in turn often break down the moral status of the individual and make him unreliable and untruthful. Only inadvertently or at second hand does the physician sometimes gain the required information, but justifiable suspicion once aroused, he can usually go to the root of the matter.



In neurasthenic, hypochondriac, and hysterical cases frequently the patient has most seriously overestimated some such habit, is morbidly apprehensive as to its results, exaggerates its every relation, and the entire life seems to revolve around this central erroneous idea. One must avoid being led by such unfortunates into adopting their point of view. The details, duration, and probable effect of the habit being clearly understood, its morbid influence can be properly estimated. Let it never be forgotten that many of these habits are symptoms, not causes, of nervous disturbance, and are the result of an underlying predisposition upon which they are grafted. There they take on a morbid development and, in turn, no doubt, add to the imbalance of the individual.

**The residence or habitat** of the patient is an important consideration. It calls attention to the climatic and local conditions favoring health or disease. Unhealthful surroundings are of immediate concern, and throw a strong light upon the causation of many nervous disorders.

**Occupation.**—Many occupations predispose to nervous maladies and sometimes furnish the cause. Indeed, a group of diseases is known as occupation neuroses, of which writers' cramp is a type. Workers in metals, particularly those handling lead, phosphorus, mercury, and arsenic; people subjected to illuminating gases or bisulphid of carbon, and those who deal in strobilites, and who are thereby likely to over-indulge, are subject to neuritis and associated mental disturbances. Work requiring exposure to cold and conditions favoring rheumatic processes entail a tendency to cerebral arterial mischief and peripheral palsies. Divers and those working in caissons, or elsewhere, under increased atmospheric pressure frequently present spinal lesions with paraplegic symptoms. Occupations which demand constant mental strain and sedentary habits, as in speculative mercantile life, teaching, and some of the professions, furnish large numbers of neurasthenics. On the other hand, the unoccupied are likely to become selfish, introspective, hysterical, and hypochondriac.

**The illness.**—From the patient's statement, his personal history, and the physician's observation, the noting of the details of the illness under consideration is often a simple matter. The medical man from his special knowledge must supplement the impressions of the layman. In the "anamnesis" preceding ataxia he discovers initial features of tabes, and in some long-antecedent moral shock he recognizes the origin of the fixed hysterical idea that may have eventuated in a contracture. For each major group of nervous maladies, psychic, cerebral, spinal, and peripheral, he must follow out the clues his training recognizes or his studies and experience suggest.

Beginning with initial symptoms and alleged, suspected, or positively known causal conditions, the manifestations of the disease are to be systematically, briefly, and clearly developed and noted, with full attention to remissions, intermissions, or relapses. In convulsive disorders a full clinical investigation of the attack is of prime importance. It should embrace its exciting cause, onset, features of consciousness, motor signs and phenomena, attitudes, duration, termination, and

sequence. In sensory disturbances investigate the particular dysesthesia or pain, its onset, exact location and outline, its intensity, duration, and associated conditions. In paralytic maladies determine the mode of onset, exact distribution, and the character and degree of motor failure. The mental symptoms are to be noted with equal care, attention being directed to their fixity, emotional character, and reasonableness or complete opposition to facts within the patient's range of knowledge. An important question is whether the patient can correct his mental ideas by mental effort or is entirely subjugated by his delusional states. Finally, the tendency to improvement or the reverse should be indicated.

**Physiognomy of the Patient.**—Formerly it was considered sufficient to describe a patient as of the nervous, lymphatic, or sanguine temperament, and this has a certain value, but a better conception of the physical status is to be had from a study of the physiognomy of the individual. Based upon embryological conditions and formulated with some precision by Lombroso, Dejerine, Weismann, Féré, and others, we can recognize a type of degenerate or, preferably, defective individuals, from whom are largely recruited the neurotic, the insane, and the criminal classes.

The marks of this type are called the *stigmata of degeneracy*, and may be divided into the morphological and the functional.

**Morphological Stigmata.**—Commencing at the crown of the head, the *scalp of hair* at the vertex which normally is close to the median line may be widely displaced or duplicated. The *cranial configuration* is often abnormal in outline, capacity, or dimensions. The occipital protuberance and ridges, the frontal eminences, and the mastoid processes are usually excessively developed. The *facial angle* is reduced, the contour of the face asymmetrical, the *lower jaw* disproportionately large and prognathic. The *hard palate* is sharply vaulted, the *alveolar arches* narrow, disproportionate, saddle-shaped, or angular and badly articulated; the *teeth* defective, misplaced, with sometimes persistence of milk-teeth late in life. The *ears* are disproportionate in size, misplaced, malformed, particularly at the root of the helix, which may bifurcate, or the tragus and antitragus are misplaced, while the *concha* is crumpled or has a tendency to stand out widely from the head. The *eyes* show notable defects, extreme refraction anomalies, squints, different colored irides, and disproportionate lids and palpebral openings. *Deviation of the nose*, *septal deformities*, *harelip*, *slit palate*, remnants of *branchial clefts* in the neck or in front of the ears, and the presence of other anatomical deficiencies are frequent in this class of persons.

On the part of the trunk, *spinal humps*, *several growths of hair*, *deep sternal furrows and scars*, or *disproportion between thorax and abdomen* are to be noted.

**The Extremities.**—The upper and lower limbs may be disproportioned to each other or to the trunk. They may be unequal in length and development. The *hands and feet* may be too small or too large. There is often a tendency to left-handedness and left-sided overdevelopment. *Deformities of the fingers*, such as *syndactyly*, *polydactyly*, *deviations*, *distortions*, *excessive length or shortness*, especially undersize of the



ring and little fingers as compared with the rest of the hand, are common in degenerates.

The genitalia in the male, besides a general lack of growth, are frequently developmentally defective, presenting hypospadias, epispadias, extrophy of the bladder, cryptorchidism, congenital phimosis, scrotal fissure, etc.; while in the female, imperforate hymen, double vagina and uterus, and hypertrophied clitoris and labia are not rare.

Taken *as a whole*, the degenerate physique is often marked by a diminished stature and an inferior vigor. Many neurotic males present the general body-conformation of the opposite sex, including sloping, narrow shoulders, wide hips, excessive pectoral and pubic adipose deposits, with a lack of masculine hirsute and muscular marking. The female may present masculine characteristics, and in such case the opposite sexuality may be further manifest in the actions, dress, manners, voice, and mental qualities of the individual. Both sexes may retain the physical attributes of childhood,—*infantilism*,—and in these cases the mental development is always retarded.

On the part of *the skin*, albinism, melanism, and multiple nevi are sometimes degenerate accompaniments. A general lack of thorough development in the dermal structures is manifested by defective hair and nails and simplicity in the papillary lines of the finger-tips.

The functional stigmata of degeneracy show themselves: (1) *Mentally*, in defective mind qualities. These vary in degree from idiocy to simple retardation of speech development, in aberrant mental and moral tendencies, among which may be enumerated destructiveness, wilfulness, indecency, deceit, and sometimes extreme acuteness and even perversity in limited fields. Genius is essentially abnormal however valuable it may be to the individual and to the race. It is often attended by many of the physical stigmata of defect.

(2) *Physically*, may be mentioned awkwardness in walking, stammering, incontinence of urine, incontinence, color-blindness, deaf-mutism, perverted tastes, and cravings leading to alcoholism and other stimulant addictions. Perversions of the genic sense, marked by sexual crimes and delirious practices, are also common. Degenerates have frequently a lack of adaptability to their environment, and so more or less strongly depart from the type and tend to extinction, subjugated by the law of survival of the fittest.

In estimating the various marks of degeneracy it is clear that very few of them, taken alone, would justify the classification of their possessor among the defectives, and it is true that a very great many of the minor stigmata may be present in a given case, associated with strong mental, moral, and physical attributes. All of them, from cleft palate to moral imbecility, are referable to defective development. However, in the presence of numerous indications of physical defect we are entitled to expect the association of their mental and moral analogues. Hence their importance to the neurologist.

The mental condition of the patient should not be overlooked. Disturbances in the psychic sphere are very common in nervous disorders and often overshadow them. Persistent depression or excitement



out of proportion to their causes, and *delusions* and *hallucinations* that may or may not be properly recognized and corrected by the patient, require close scrutiny. Especially in hysteria are we confronted by a train of mental symptoms, attitudes, and reactions that may easily be confounded with insanity or which actually carry the patient over the rather broad, dividing neutral ground into the realm of alienism. *Loss of self-control, irritability, increased emotionality*, and *vague or formulated apprehensions* are the ordinary concomitants of neurasthenia and psychasthenia. Many cerebral diseases produce *incoherence*.

**Sleep** in nervous patients is one of the most important practical considerations. If it is disturbed, seek the cause, remembering that habits of wakefulness are easily formed. Distressing, formulated, and repeated *dreams and nightmares* are the neurasthenic's portion and the expression of his lowered nervous and physical tone. *Somnambulism*, nocturnal urinary *incontinence* and *night-terrors* are the common property of nervous individuals in childhood. Some patients find difficulty in falling asleep, others in remaining asleep; others are simply unrefreshed upon awaking. The selection of specific remedies and the time of their exhibition turn upon such considerations.

**Memory.**—Nearly every nervous invalid asserts a *loss of memory*, which rarely, however, exists. This mental faculty varies not only greatly in individuals, but is subject to great modifications in a given individual under different conditions of health and age. To the keen perceptions of a child everything is novel, is deeply imprinted in the mind, and is rarely forgotten. Later in life a new face or name is no rarity, is not sharply apprehended, and its recollection is consequently difficult or impossible. The old, in part for this reason, remember their early experiences better than more recent happenings. In physical ill-health and in conditions of mental distraction or introspection, as in hypochondria, hysteria, and neurasthenia, the alleged loss is really a lack of the mental concentration that constitutes the essential basis for good memory. In such cases this may be demonstrated by a few questions on remote personal happenings, which will usually be recited with extreme minuteness and detail. A loss of memory may embrace a certain definite period of time. When this occurs as a result of head injury or the action of some of the poisons, usually the carbon group, it may extend for some time anterior to the cause, as well as for a period following the cerebral accident. In many delirious conditions the patient recalls his experience vaguely or in a dream-like manner.

## CHAPTER II.

## THE GENERAL PHYSICAL EXAMINATION.

**Present Condition.**—What has gone before prepares the way for a thorough physical examination. Whenever possible, the clothing of the patient should be entirely removed, as study of the physical human outlines is most valuable. Without this step spinal deviations, chest deformities, lack of symmetry in the limbs, or other serious defects of the most important diagnostic character may escape notice. Upon sketchy outlines of the human figure supplied in text-books and by dealers abnormalities of form and function may be indicated with precision. No lesion is too slight to be overlooked, and no assertion of functional health is to go unquestioned. Remote conditions are not infrequently caused of central disturbances, and central mischief is manifested by peripheral states. The nutritive process may first engage attention.

**The Alimentary Tract.**—The condition of the *tongue* in relation to maturation and abnormities of position or evidence of inherited syphilis, the color of the gums with reference to anemia or evidence of metallic poisoning, such as the blue line of lead and the sponginess of mercury and phosphorus, can be noted at a glance. Any infection of the gums, tonsils, or nasopharynx requires especial observation. Particular attention is to be directed to the *larynx*. Aside from indicating the state of the stomach, it may give important evidence of nervous diseases. The fine fibrillar twitching of general paresis and bulbar palsy, the tremor of alcoholism, the contortions of chorea, the lack of motility and atrophy in lalloglossolaryngeal paralysis, and the deviation on protrusion in hemiplegia are positive signs of great value. Very often the patient's *breath* furnishes information. Alcohol, various drugs, including mercury and some systemic diseases, notably diabetes, give peculiar odors to the expired air. Catarrhal and pyemic states of the mouth and nasopharynx are usually attended by some *fætor* of the breath. Difficulties in swallowing are very significant. Abnormalities of *appetite* for food, gastric and intestinal indigestion, constipation, and *costal conditions* are significant in many ways. Attacks of *colic*, *recumbency*, *diarrhea*, and *rectal hemorrhages* have special bearing on the condition of the reflex spinal centers. The condition of *spleen* and *liver*, as in acute and malarial infections and chronic alcoholism, may give important indications of constitutional and local states that have a relation to the nervous phenomena under investigation. Displacement of the abdominal viscera, such as *enteroptosis*, is considered very important in certain cases.

**The Respiratory Organs.**—In the *nasal* and *pharyngeal spaces* inflammations, new growths, or irritation zones may furnish the starting-point for morbid states of the most varying nature, as hysterical sneezing, spasmodic asthma, and pronounced neurasthenia. *Laryngeal* and *pharyngeal polyps* and *excesses* require a careful topical examination. In the condition of the *lungs* and *pleura* we seek for local explanation of various symptoms such as respiratory pain and oppression, *costal*



neuralgia, continuous cough, or for the evidence of tubercular deposits, explanatory, perchance, of a cachexia that might otherwise be erroneously referred to nervous depression.

**Circulatory Apparatus.**—The condition of the heart is revealed only by a thorough physical examination of its position, size, action, and valvular competency. The condition of the arteries, patent to the eye in a tortuous temporal, to the finger in radial atheroma, should be still more extensively investigated in the femoral, brachial, carotid, and other superficial regions. The condition of the *blood-pressure*, as shown by arterial tension, on the two sides of the body, near the heart, and at a distance, is worthy of careful study. It enables one to draw analogical conclusions as to the circulatory apparatus of the central nerve-organs. The sphygmomanometer is to be employed in all cases of circulatory disturbance. The condition of the arteries is the best index of the real age of an individual. In them we often find evidence of a premature decay out of all relation to the years that have been lived. Sphygmographic tracings, as a method of record and precision, have their own value. The *pulse*, by its lack of rhythm, particularly by a tendency to great variation in its rate, depending upon slight exciting causes, often shows the unstable nerve-tone of the patient or a general isothemia. *Fluoridæ*, *morbidæ*, local *crasis* and *crises* are vital expressions of angioneurotic disturbances. The blood must be examined for parasites, hemoglobin, and corpuscular conditions. The severe anemias have a very important relation to brain and spinal symptoms. A marked *leucorhœgia* attends inflammatory and purulent processes affecting the brain and spinal cord. The presence of cholin in the blood is frequent in diseases marked by degeneration of nervous tissues and in epilepsy. Of very great diagnostic importance is an examination of the spinal fluid obtained by spinal puncture. The various bacteria associated with inflammatory processes may thus be demonstrated, and in degenerative conditions of brain and spinal cord, especially in general paresis and locomotor ataxia, a marked increase in the cytological contents of this fluid and definite globulin changes are almost invariably found. The examination of both blood and spinal fluid by the Wassermann and other methods is essential in any case where syphilis is a possible etiological factor.

The **temperature** may be greatly modified by nervous diseases. Organic brain-lesions may upset the balance between the thermocætic and thermogenic centers, producing either a very high or a markedly subnormal body-heat. In hysteria a very high range of temperature is sometimes noted without the usual concomitants of fever. In cerebral hemorrhage, basilar meningitis, and brain tumor the temperature is often below the normal. Early in cerebral hemorrhage the paralyzed side presents usually a disproportionate elevation of a degree or more of heat over the opposite half of the body, as is shown even by axillary temperatures. The temperature of the paralyzed side later becomes sub-normal. Slight variations of the central normal temperature, usually in an upward direction, are frequently observed in pure neurasthenic states, while the extremities are commonly cold.

**The integument.**—From the appearance and condition of the cutaneous expanse much is to be learned as to the general health of the

individual and the activity of his physical functions. The skin may be greatly modified by nervous maladies. In some instances the dermal manifestations make up the major part of the disease, or the dermatosis may be an associated feature of other neurotic disturbances. All varieties of *urticaria* are of frequent occurrence among the neurotic. *Derмоgraphia* and the *trache ciliaris* of meningitis demonstrate the vasomotor irregularities. *Herpes* and *eczema*, limited to the anatomical distribution of nerves or spinal segments, as in *zoster* on the face, trunk, or limbs, declare the nervous involvement. *Nevi* are apparently related to the nerve distribution in many instances. Chains of neurotoma are beaded along peripheral nerves with or without cutaneous discoloration. Neuromas of long standing are frequently marked by dermal changes of increased or decreased nutrition, as witness the thickening of the skin of the face in neurinoma of the fifth cranial nerve and the subsequent flattening or the actual loss of the eyebrow and hair. The neuritides, if of a severe grade, show dermal dystrophy as well as muscular wasting. The epithelial structures involved may take on increased growth if vascular stasis favor increased nutrition, giving rise to scaldiness of the skin and increased growth of the hair and nails. More frequently atrophic changes follow; the skin is thinned and glazed, the epithelium scant and poorly protective of the more highly organized subjacent tissue. The hair becomes dry, brittle, and sparse, and the nails rough-ridged and sometimes covered with overlapping scales. *Pigmentary changes, swelling, and blue edema* are not infrequent in hysteria. The enormous *thickening* in myxedema and acromegalia are also due to perverted trophic control. The *redness* of erythromelalgia and the *opacified appearance* in Raynaud's disease are striking features.

**Genito-urinary Tract.**—In the genito-urinary tract are found many conditions bearing an intimate relation, both causal and symptomatic, to nervous diseases. Some of them are overlooked or unknown to the patient, and others receive altogether too much attention at his hands. A thorough clinical examination of *the urine*, which should be quantitative as well as merely qualitative, is best made from a sample of a carefully measured twenty-four-hour collection. It shows at once the eliminative powers of the organism through the important excretion outlet of the kidneys. A loosened output of *uric*, or the presence of *albumin* or  *sugar*, give important data as to the blood-state and may explain grave cerebral manifestations, such as convulsions and coma. A very *low specific gravity* is noted after hysterical attacks. A large quantity of *phosphates* and *urates* is common to many neurosthenic conditions, and an *overconcentrated urine* is usual in purtic states of the detrusor urinae. After an epileptic attack the specific gravity and solid constituents of the urine are increased. An excess of *acidica* is found in many nervous conditions attended by inefficient elimination.

The microscope, besides giving evidence of organic disease, such as nephritis, proctitis, and cystitis, may show spermatozoa from a relaxed control of the outlets of the seminal vesicles, but more often demonstrates that the deposit considered seminal by the patient is devoid of testicular products.

Anuria in nephritis is of most serious import, though it may exist



almost indefinitely in hysteria when associated with persistent emesis, whereby the uric products are vicariously ejected.

The state of the *bladder* is made out largely by the investigation of the urinary secretion, but the bladder and *ureters* are open to more proximate methods. The size, expulsive and retentive powers of the bladder, its contents, and the condition of its mucous lining are, in suitable cases, to be investigated with precision. Loss of *sphincteric control* of the bladder in paraplegia and ataxic conditions is usual, while in meningitis, and in some atonic states generally, *retention* of urine is to be expected and provided for.

The *external genitalia* rarely give much information. In males *prostatitis* adhesions and accumulations or a long *phimotic prepuce* may be the source of irritation and the inciting cause of general nervous phenomena. Here, as elsewhere, any abnormal and correctable state should not escape appropriate attention. This is emphatically true of the *degenerating generative organs* in the female. Undoubtedly undue importance has attached to them and much ill-adviced meddling has been bestowed, but a lack of integrity on their part should certainly engage methodical treatment.

The *genital sense* is usually blunted or completely destroyed in advanced locomotor ataxia and spinal lesions which cut off peripheral sensation. It is reduced in all depressed physical states, whether associated with marked nervous phenomena or not. On the other hand, the genital reflex may be accentuated in lateral sclerosis of the cord, and priapism, unattended by increased desire, may be a troublesome feature. In injuries to the cervical portion of the cord, priapism is likewise common.

The question of sexual irritation and overindulgence calls for more than ordinary thought. Excess is a matter purely relative to the individual and his condition at the time of indulgence. Overuse of any organic function is shown by persistent fatigue and irritable prostration. This may here furnish as a working criterion, but we are to remember that matters have already gone too far when the great margin of natural reserve power has been overdrawn and even temporary debility produced. Thus, an amount of masturbation or sexual indulgence insignificant in a sturdy individual, is sufficient in the defective neurotic to induce a profound depression.

**Spinal puncture**, after the manner of Quincke, is a very important diagnostic procedure in many diseases of the brain and spinal cord. The fluid so obtained may show bacteria of infection and pathogenic characters; *lymphocytes* and blood-cells in inflammatory and hemorrhagic conditions; *epithelium* in syphilis; marked increase in cellular elements, especially the lymphocytes in locomotor ataxia and general paralysis, and in these two conditions the Wassermann and globulin tests are often positive in the spinal fluid even when the blood gives a negative reaction. In some spinal tumors the fluid presents a very remarkable *yellow color*. In spinal and even in cerebral hemorrhage it may carry a decided admixture of blood. Spinal puncture is a dangerous procedure in brain tumor, and should then be employed only with great caution, especially in neoplasms of basal location, owing to their tendency to be forced down upon the medulla by the withdrawal of the spinal fluid support.

The amount of intraspinal and intracranial pressure can be roughly inferred from the force of the escaping stream. It can be accurately measured by attaching glass tubing to the puncture needle and noting the height to which the fluid mounts in the vertical tube. Various manometric devices are also available for precise observations.

### CHAPTER III.

#### THE MUSCULAR SYSTEM.

**Motility.**—It is a rule with hardly an exception that nervous disorders are marked by *errors of motility*. These vary from a condition of slight general weakness, or *parosis*, to complete loss of muscular power, or *paralysis*; from slight *tremor* to rigid *contractures*. The character and distribution of the muscular difficulty is often of the first importance in diagnosis and in localizing limited lesions.

The *station, attitude, and gait* of the patient, depending as they do largely on muscular force, control, and activity, often furnish most important information. The *contractured, semiflexed position* of the upper extremity and the *rigid lower limb, swung en avion* from the pelvis with dragging toe, mark the *hemiplegic*. The bowed and trotting



Fig. 1.—Matison's hand-dynamometer fixed with double side levers.

*gait of paralysis agitans*; the stamping and sprawling of *tubes dorsalis*; the stoppage or high knee-action with dangling feet that results from multiple neuritis; the spastic, rigid, and trembling legs of paraplegia; the dangle-leg of anterior poliomyelitis; the sweeping gait of hysterical leg paralysis; and the cerebellar stagger tell their own story. Minor peculiarities are no less, but rather more, important. The spinal rigidity and constant guarding against jars that dominate the attitudes and gait of the subjects of spinal injuries; the distorted features of cranial-nerve palsies, athetoid movements, chorea; twittings, restlessness, slight limps, awkwardness; attitudes of limbs, trunk, or head long or customarily maintained, have one and all a significance that cannot be overestimated.

**Reduced Motility.**—For testing muscular strength numerous recording instruments are available. In the hand-dynamometer of Mat-



here we have a simple means of testing the grasp, and by fitting it with light handles attached at either end it can be employed in various ways to test the muscular groups of the upper and lower extremities. The examiner always has in his own person a check upon the records of the patient. When one handle is attached to a book in the floor, the lifting power of the back, the legs, or the arms is readily ascertained. Readings on such an instrument are of the greatest value for clinical records and as a means of comparison at future times.

But without any such instrument the physician can roughly test every group of muscles by opposing the efforts of the patient. To examine flexor and extensor muscles of the wrist, elbow, and shoulder, instruct the patient to resist your efforts to communicate motion to the respective joints. The same can be done in the lower extremity with the ankle and the knee. The hip is tested by having the patient mount a chair, ascend stairs, and raise the limb to a horizontal position. Both hands of the examiner grasped by those of the patient as vigorously as possible enables the physician to compare their strength.

In hysterics and malingerers it is sometimes difficult to feel that full efforts have been put forth, and in other instances muscular exertion is inhibited by pain or tenderness. In these cases particularly, the muscular tests may be advantageously varied and repeated by securing a large number of movements, such as having the patient stand on one foot, rise on the toes, stoop, crouch, lift chairs, and execute other tasks when he is not conscious of the purpose in view. Small movements of the fingers are very clearly studied as the patient buttons his clothing or handles different objects, which may, with design, be placed in his way.

Where the degree of feebleness is slight, it perhaps is manifest simply as fatigue after exertion or in walking a comparatively short distance. In some cases muscles which at first register considerable strength are rapidly exhausted by a few full contractions. In others initial efforts are weak, but quickly increase to a normal power, making it needful to always take an average of several tests.

The limits of the paralysis or paresis must be precisely noted, whether confined to a single muscle or muscle group, to the distribution of a single nerve or group of nerves, to a single limb, to the face, to one side of the body. If the involvement is widespread, it will probably vary in degree in different regions, and this is also of importance. The terms *monoplegia*, *hemiplegia*, *paraplegia*, and *diplegia* are employed respectively as the face or a single extremity, the lateral body-half with the corresponding limbs, both lower extremities, or both sides of the body are involved.

Not only is it necessary to record the strength of the muscles under examination, but also the manner in which they perform. For all precise movements, complete balance of muscular action—*synergy*—is a prerequisite. If the flexors of a joint are weakened, the extensors not only fail to execute their function with exactness, but their strength is also diminished and the resulting volitional movement is weak or clumsy in consequence. In conditions marked by errors of sensation, especially by impairment of the muscular sense which gives a knowledge of the position of our members and enables us to estimate weight, movements



become uncertain. This uncertainty is generally exaggerated if the movements are not guided by the eyes. *Incoordination of movement* results. This is sought for, and, if present, is demonstrated by having the patient, with closed eyes, touch given points, either on his own person or elsewhere, as by bringing the index tips of both hands together, touching the tip of his nose, the lobe of his ear, or the finger of the examiner. In the lower extremity we ask him to touch a given object with the point of the foot or reproduce with the other the position given a limb by the examiner. In making this last test it is necessary to avoid furnishing the patient such information as might come from contact with the bed or personal clothing. Standing with the eyes closed and the feet together, reducing the base of support, is often attended, in spinal and other nervous diseases, with swaying of the body and a tendency to fall. This constitutes *static ataxia*, and is known as the Romberg sign. It may be graphically recorded by attaching a marking-point to the patient's head, which traces his ataxic movements on a prepared surface held at the proper level. Having the patient walk backward with closed eyes or stand on one foot increases the ataxia.

Incoordination may be very great, so that the blindfolded patient does not come within several inches of his nose in his attempts to touch it with his finger, and has no certain knowledge of the position of his limbs under the bed-covering except as he informs himself by sight. While standing, he may fall at once if his eyes are closed, or even reel in his chair.

The following example of ataxic writing shows incoordination very clearly, and indicates another important test of muscular balance:

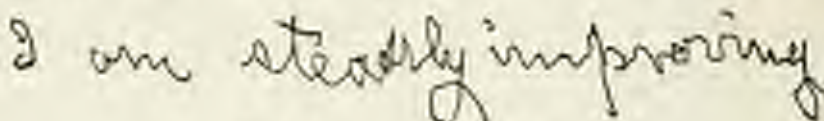


Fig. 2.—A specimen of ataxic handwriting.

**Increased Motility.**—Perhaps motility in nervous disorders is more frequently excessive than deficient—that is, beyond the purposes of the patient. Parietic conditions, too, are not infrequently associated with irregular and involuntary muscular movements.

*Tremors* are the accompaniment of overexertion and emotion in health, but often become significant symptoms of nervous disturbance and disease. A tremor that exceeds six to the second is called *rapid*, one below that rate *slow* or *asthenic*, while the amplitude of the muscular vibration or excursion is indicated by the adjectives *fine* and *coarse*. Tremors occur sometimes during complete rest, as in paralysis agitans, or only when the involved muscles are called upon to act—the “*volitional*” or “*intention*” tremor, as in multiple sclerosis. While tremors may involve the entire muscular system, as is often the case in exophthalmic goiter, they may be restricted to a single limb or group of muscles, to a single muscle, or even to a few muscle fibrils. This last constitutes a *fibrillary tremor*, or, rather, fibrillary spasm, because it is usually devoid of rhythm, and presents only one or a few irregular twitchings under

the integument. They may sometimes be provoked by stroking or tapping the surface or putting the muscle into operation.

Edner,<sup>1</sup> as a result of an extended series of experiments, concludes: (1) All muscular movements are made up of a series of elementary contractions and relaxations, which may be appreciable as tremors in conditions of both health and disease. (2) The differences between different tremors are of degree rather than of kind—i. e., no form of tremor is distinctive of any one disease or group of diseases. (3) No definite relation exists between one form of tremor and any other. (4) The frequency of movement is in inverse ratio to the amplitude and vice versa. (5) Habitual movements are performed with greater freedom from tremor than unusual movements. (6) There is no material difference between the movements of the two sides of the body, except as related to proposition 5.

In *tapping for tremor* the patient is directed to stretch out the arms with the fingers extended and separated as widely as possible. The difficulty becomes at once apparent, or is felt as a thrill by the examiner's hand grasping the wrist or placed against the finger-tips of the patient. Care must be exercised not to mistake the origin of the tremor, as vibrations of the head and extremities may be communicated from a distant point. Again, tremor in the tongue and face is best shown when the patient, with closed eyes, protrudes the tongue vigorously for a few moments. Intention tremors are usually best shown when the patient attempts to carry a full glass of water to the lips, to write with a pen, or to execute other delicate coordinate movements. By means of registering apparatus a tracing of the tremor may be obtained in a graphic manner, and for this purpose the sphygmograph may sometimes be employed. The following specimen of writing in a case of paralysis agitans most clearly shows the amplitude, rhythm, and, by timing the effort, the rate of the tremor:

John Erickson

Fig. 3.—Specimen of handwriting showing tremor of paralysis agitans.

**Spasm.**—When a muscle or group of muscles contracts more or less energetically without the conscious intention of the individual, it constitutes a spasm, and is *clonic* or *tonic* as it is frequently repeated or steadily continuous. A painful spasm is usually called a *cramp*, though a facial spasm, accompanied with pain, receives the now classical designation of *tic douloureux*.

Long-continued spasm leads to *contracture*, a condition in which the muscle eventually becomes structurally shortened. Contractures follow, also, through natural tones, when a muscle is for a long time unopposed by its physiological antagonist. *Postleptic movements* sometimes follow brain-lesions of long standing. The paralytic portions of the body manifest peculiar involuntary movements, which may vary from slight twitchings of a choreic character to the never-ceasing, purposeless activity

<sup>1</sup> Jour. Exper. Med., 1897.



shown in athetosis, where the fingers and toes work in a tentacle-like, or amoeboid, manner. Voluntary efforts usually increase these athetoid movements. In hemiplegia, at times the paralyzed members duplicate the voluntary motions of the sound side, producing associated movements. They are particularly likely to occur under instinctive action, such as stretching and yawning, or under strong voluntary efforts with the sound side, as in the attempt of a right-handed, right-sided paralytic to write with the left hand.

*Convulsions* consist of more or less widely distributed, persisting or repeated spasms, associated usually with disturbed or abolished consciousness. They are symptomatic of numerous cerebral disorders, injuries, and intoxications. We speak of them as being *general* when the entire body is affected, or *local* when the convulsive movements are confined to a limited portion of the muscular system, as the face, hand, or lower extremity. Local convulsions are frequently styled *Jacksonian*, especially if marked by progressive extension to adjacent muscles, with tardy, slight, or no involvement of consciousness. The initial spasm or sensation of such fits is called the *signal spasm*, indicating fairly the cerebral center in which the muscular storm arises. Convulsions are *acute* or *chronic*, as the spasms of which they are made up are long maintained or frequently repeated.

The careful investigation of a fit, and the importance attaching to its details, their order of appearance with all associated, precursory and sequential phenomena, have already been touched upon. When these clinical facts must be learned from lay persons even of the most intelligent character, the difficulties are often insurmountable. In some cases a trained medical man or a well-instructed nurse must remain with the patient for the purpose of getting a complete description of the attack.

In rare cases the ordinary muscular tone is abnormally increased—*apostolic*. This gives rise to a condition analogous to a tonic spasm, and voluntary effort is thereby delayed, as in Thomsen's disease, and to a lesser degree in paralysis agitans. *Hypertonus* and *hypotonus* of the muscles with corresponding increase or decrease of tendon reflexes are presented by spinal and cerebral lesions.

**Reflexes.**—In the muscular reflexes we have a series of signs which give information regarding both the spinal centers, and the nerve-paths above and below these centers. They have great diagnostic value and localizing importance. A knowledge of the spinal-cord segments to which they belong, and of the anatomical relation of those segments to the vertebral bodies and spinous processes, with the association of muscle and sensory areas, forms the basis of spinal localization.

A *muscular reflex action* is the result of a peripheral stimulation reaching motor spinal centers and thence centrifugally manifest in a contraction. Thus, in the pupil, the stimulation caused by light falling on the retina travels by the sensory limb of the reflex arc to the medullary center, and there calls forth energy which flows down the motor limb and causes pupillary contraction. A blow on the patellar tendon stimulates centripetally the lumbar center, and a contraction in the extensor muscles of the leg results in the "knee-jerk." The normal



activity of the reflexes requires not only the integrity of the center and both limbs of the reflex arc, but a proper association of the center and the higher-lying cerebral levels. Any block or interruption in either limb of the arc, or the destruction of the spinal center, abolishes the reflex. At the same time it is a general rule that pathological conditions interfering with the free communication between spinal center and cerebral cortex tend to increase the reflex manifestations of the cord. Monakow<sup>1</sup> points out that in brain lesions the hypertonus and reflex excitability are more marked the nearer the lesion approaches the spinal cord, and Bergman<sup>2</sup> shows that a cortical lesion may be entirely unattended by increased reflexes. In man, however, the absolute destruction of all such communication—in other words, complete division of the cord—produces abolition of muscle reflexes below the level of the lesion. Sensitiveness in the part tested is ordinarily attended by an increase of reflex irritability, as, for instance, in rheumatism.

Reflexes have been variously described as skin, superficial, deep, tendon, muscular, and organic,—distinctions that have no especial clinical value. Many of them can be inhibited voluntarily by the patient, and all require complete passivity on his part for their proper investigation. Some only appear as a result of disease. As a rule, the muscle under examination must be slightly stretched, and then the blow or passive motion, which suddenly increases its tension, gives rise to the reflex movement.

The contact or threatened contact of any object with the eyelids or conjunctiva causes a *palpebral reflex*—the closing of the eye.

A *open-ocular reflex* has been described by McCarthy. It is elicited by percuting over the supra-orbital nerve either at the point where it emerges or somewhat higher up in its field, and consists of a fibrillary twitching of the orbicularis palpebrarum. It should not be confounded with the palpebral reflex. It is absent in severe injury or disease of the facial and of the fifth cranial nerves.\*

The *pupillary reflex* is manifest under several conditions: (1) If the eyes have been closed for a few moments and then suddenly opened in a strong light, or if in a dark room a beam of light is thrown upon them, the dilated pupils quickly contract. (2) The normal eye shows a decided pupillary contraction if focused on a near object—say, at eight inches—after being directed at a distance of over twenty feet. This latter is sometimes called the *constrictor reflex*. These normal reactions are variously modified by disease. They may be simply sluggish, they may be entirely absent, or they may be dissociated. In locomotor ataxia and in parietal dementia, when the posterior portions of the cervical cord



Fig. 4.—Method of eliciting the jaw-jerk.

<sup>1</sup> "Göttinger Pathologie," 1908, II. Auflage.

<sup>2</sup> "Brain," 1910.

<sup>3</sup> "Neural Centralbl.," Sept. 1, 1901.

are sclerosed, the pupils usually are narrowly contracted. They then respond to efforts of visual accommodation only, and are quite insensitive to light. This is known as the *Argyll-Robertson pupil*. Rarely the opposite condition has been noted in other diseases. The action of certain drugs on the pupil, as opium and belladonna, is always to be kept in mind when looking for this reflex, and the eyes must be separately observed, as only one may be affected. The patient must not be allowed to close the eyes vigorously in testing for the pupillary reflex to light, as this not causes the pupils to dilate, though covered by the lids. In certain conditions, when a portion, usually one-half, of the retina is blind or insensitive, a narrow ray of light made to fall upon this part of the optic nerve, by being thrown obliquely through the pupil, causes distinct contraction. Wernicke first pointed out that this occurred only when the lesion causing hemianopia was situated back of the geniculate bodies in the optic radiation or in the visual centers, and the condition is known as *Wernicke's sign*. (3) Sharp pinching of the skin on the side of the neck and severe pain in general cause a dilatation of the pupil.

The *jaw-jerk*, or *masseteric reflex*, is produced best by placing a ruler or similar object on the lower incisor teeth while the mouth is



Fig. 5.—Method of eliciting the jaw-jerk.

partially opened, and striking down upon it with a percussion hammer. This is followed by a quick contraction of masseter and temporal muscles, and usually by the active closing of both eyes.

The *pharyngeal reflex* is the spasmodic action, familiar to every one, produced by tickling or stroking with any object the faucial pillars or the walls of the pharynx, and sometimes even the soft palate or base of the

tongue. It is commonly absent in hysteria. Patients who have been subjected to much local pharyngeal treatment sometimes acquire much tolerance of manipulation and control this reflex completely.

In the upper extremities there are a large number of muscle reflexes which may be elicited by striking on the insertion tendon after so placing the member that the muscle under investigation is put in moderate passive tension. Unless the patient completely relaxes, it may be quite impossible to demonstrate the reflex, and it is often difficult to secure this relaxation even in well-intentioned persons, unless their attention is distracted.

The various portions of the *deltoids*, *pectoralis major*, *trapezius*, *ser-*



*reflexa magna*, and *scapular muscles* may be made to react in this way under the percussion hammer in most persons, but are much more active in neurotics and in those spinal diseases marked by increased reflexes. With the arm extended and supinated, a tap at the insertion of the *biceps* causes a flexion movement at the elbow. The *flexors of the wrist and fingers* similarly respond while the extremity is in this position. A tap on the lower end of the radius actuates the *supinator longus*, giving in marked cases a flexion movement of the elbow.

With the elbow flexed, as by allowing the arm to hang over the back of a chair, the *biceps* is actuated by a tap above the point of the elbow. With the forearm slightly pronated and the hand hanging, as in the usual position on a chair-arm, the *wrist and finger extensors* can be similarly examined. Of the small muscles of the hand, only the *polevris brevis* can readily be brought into play in health. This is done by making pressure over the pisiform bone and lower end of the ulna with the thumb and forefinger, and causes a grooving in the ulnar border of the hand just above this point.

In some spastic cases the passive sharp flexing of fingers and wrist may develop a *spasmodic*, consisting of rapidly repeated movements of extension and flexion at the joint, which tend to persist as long as gentle tension upon the extensors is maintained by the examiner.

On the *dorsum of the trunk* there are a series of reflexes which, below the scapulae, are not of much diagnostic value, and which can usually be demonstrated by stroking, pinching, or, preferably, percussing the muscular masses. Anteriorly, with the patient lying supine and the abdominal wall relaxed, a tap on the costal cartilages on either side of the xiphoid depression causes a dimpling or lateral movement at this point, called the *epigastric reflex*. A similar tap on the costal border in the nipple line, acting through the abdominal oblique muscle, produces the *abdominal reflex*, most noticeable at the umbilicus, which is promptly drawn toward the side percussed and in the oblique direction indicated. Sharply stroking the lateral abdominal surface with the nail or handle of the percussion hammer will frequently be found the best mode of eliciting this reflex. In this manner upper, middle, and lower abdominal reflexes can be produced. Percussion over the upper and lower portions of the rectus abdominus muscle causes a movement of the umbilicus in the corresponding direction.

In the *lower extremities* we find that a series of taps along the origin



Fig. 4.—Method of eliciting the knee-jerk and relieving it by Jendrassik's method.



of the *great gluteal muscle*, when the patient is erect or prone, are followed by contractions in corresponding segments of that muscle, and a tap near the anterior superior spinous process starts the *triceps jacobæ later femoris*. The *knee-jerk*, or *patellar reflex*, being easily examined and frequently modified by disease, is one of the most important of the muscle reflexes. It is elicited generally by having the patient cross one knee over the other while sitting. The under limb, with the knee at a right angle, should support the upper, which gently rests over it with all muscles relaxed. A smart, quick blow with an object of some ounces' weight, as with the back of a thin book, the ulnar border of the hand, or, best of all, with a proper percussion hammer, upon the patellar ligament or just above the patella, is followed by a contraction of the anterior thigh muscles, causing the suspended foot to move forward an inch or two. A percussion hammer



Fig. 1.—Method of eliciting *knee-jerk*.

at least twice the weight of the one usually employed in chest examinations should be used in doubtful cases and is valuable in routine practice. If the patient is in bed, the limb to be examined may be lightly placed over its extended fellow, crossing it at the knee, and then the blow employed, or with the patient on his side, the knees partly flexed, the same thing may be done. When difficulty is encountered in securing this response, it is to be remembered that unless the muscles are fully relaxed the patient may inhibit the phenomenon, or that it may be so slight as to escape attention. If the patient be placed on a high chair or on the edge of a table so that the legs are pendent, and at the same time his attention be diverted, the jerk may usually at once be shown. It can also be reinforced, as described by Jendrassik, by having the patient grasp some object vigorously with his hands, or by merely clenching his hands at the time the tendon is struck. In children or very nervous cases this reinforcement may be secured by directing the patient to forcibly grasp the examiner's hand. It should only be considered abolished when, thus reinforced, with the limbs unclothed, the eyes closed, and the unemployed hand of the examiner upon

the rectus femoris, no response can be detected. Similar plans of reinforcement are of value in testing other reflexes, and serve to divert the patient's attention.

In some cases, where the reflex activity is pronounced, by pushing the patella sharply downward when the limb is extended a *rectus reflex* is produced. If, upon continued downward pressure, a number of rhythmic contractions ensue and are disposed to continue, we have the *rectus clonus*. Taps over the insertions of the abductor group and over the tendons of the knee flexors, at times marked by increased reflexes, produce corresponding muscular contractions.

In conditions of *reflex* or *agrabic* *irritability*, if the knee be extended and the foot strongly flexed dorsally, a sharp tap on the upper and outer portion of the leg, over the extensors of the foot, causes a contraction of the calf-muscles, and this response is called the *foot-tap contraction*. With the foot in the same position, a tap upon the *Achilles tendon* causes an extension of the ankle-joint. If there is doubt about the presence of the *foot-tap reflex*, place the patient on a chair in the kneeling position, direct him to grasp the chair-back firmly, make slight pressure over the ball of the foot to extend the tendon and use the percussor. The early absence of this reflex in *sciatica* and *locomotor ataxia* renders its observation important. The *peroneal muscles* likewise respond when properly percussed, the foot being first turned somewhat inward to put them on the stretch.

*Ankle-clonus* or *foot-clonus* is tested by sharply flexing the foot dorsally with the knee almost extended, and consists in rhythmic movements of the foot upon the leg, caused by repeated contractions of the calf-muscles. They persist usually as long as pressure is maintained against the ball of the foot. Sometimes it can best be developed by having the patient, as he sits, place the foot beside the chair in such a way that the weight of the limb is supported by the point of the foot. The clonus movement then causes the extremity to dance.

A *peroneal* persistent contraction is occasionally found when the ends of a muscle are suddenly and passively approximated. For instance, when the foot is passively flexed on the leg, a tonic contraction of the anterior tibial muscles takes place. All other muscle reflexes are momentary, and occur under conditions of extension.

Tickling or stroking the plantar surface gives rise usually to well-known contractions of a wide-spread character, known as the *plantar reflex*. They may be confined to the flexors of the toes. Instead of flexion the toes may sharply extend, particularly the great toe, constituting the important *signs of Babinski*, found only in disordered, usually degenerative, conditions of the pyramidal tracts. A blunt wooden tooth pick or a match stick may conveniently be used in seeking this sign. In some it is elicited best by irritating the outer half of the plantar surface; in others, by a stroke across the ball of the foot; and again, in others, by a stroke under the plantar arch. Oppenheim elicits this reflex by deeply and forcibly drawing the handle of the percussion hammer or the finger ends behind the inner border of the tibia in the lower third of the leg. Gordon,<sup>1</sup> of Philadelphia, has discovered also that very deep pressure of the calf muscles will in similar

<sup>1</sup> *Jour. of Nervous and Mental Dis.*, Feb., 1907.



cases produce an extension movement of the toes, and claims that it is found in cerebral lesions when the Babinski may be absent. During the first two years of life extension of the toe is the normal response. Bechterew<sup>1</sup> describes an analogous reflex response found in similar cases induced by extending the foot at the ankle-joint and passively but vigorously flexing all the toes. This manipulation is followed by involuntary extension of toes, an upward movement of the entire foot, and in extreme cases by flexion of knee and hip.

When the skin on the inner side of the thigh, or when the scrotum is sharply stroked or pinched, the corresponding testicle is more or less actively drawn toward the pubic arch by the cremaster muscle. This *cremasteric reflex* must not be confounded with the slow contraction of the dartos tunic of the scrotum, of which it is entirely independent.

The sphincters of the bladder and bowel act reflexly upon the contact of any foreign body, and the destruction or serious impairment of these *sphincter reflexes* is attended by incontinence. The *sphincter reflex* of the bladder is tested by the introduction of a sound, noting the presence or absence of the sphincteric grasp. A finger inserted in the rectum distinctly feels the *anal sphincteric reflex*, if present. Pinching or pricking the skin about the anus causes an *anal reflex* consisting in a puckering of the perianthentous margin of the bowel outlet, and this may also be attended by a distinct sphincteric contraction.

When the corona glandis penis is pinched a contraction may be felt by the examiner in the perineum due to contractions in the bulbo-cavernosus muscles. This is called the *vicile reflex*, and by some writers is thought to be an evidence of potentia coeundi.

In general, we may say: First, that the Argyll-Robertson phenomenon, Wernicke's sign, iridoplegia, ankles-, wrist-, jaws-, rectus-clonus, and Babinski's sign are never found in health, and are valuable objective signs of central disease. Second, that the abolition of the knee-jerk never occurs in healthy persons, and that the abolition of the sphincter reflexes is strongly indicative of central disease. Third, that the abolition of the pharyngeal and plantar reflexes, with increase of others, is presumptive of hysteria. Fourth, that a moderate intensification of all reflexes frequently indicates neurasthenia. Fifth, that the abolition of all reflexes in a given anatomical area points to histological disease, either central or peripheral, in the area supplying those reflexes.

It should be pointed out that in hysteria and neurasthenia there is sometimes a *spurious make-clonus* that may be misleading, the foot making only a few vibrations upon the institution of the test. In a real clonus the repeated contractions are confined to the muscle or group of muscles related to the individual movement; in spurious clonus the antagonists are also active to some extent. In true typical clonus the movements are regular and rhythmical; in the spurious variety irregularity, inequality, and a lack of rhythm are observed. In the spurious condition, too, there is a tendency for the reflexes to be widespread, so that a tap upon one patellar tendon may cause both legs to respond, start the upper extremities, or almost convulse the patient.

*Myoclonus*, or *shock-jitter convulsive spasm*, is produced in certain irri-

<sup>1</sup> "Neuro-Central," No. 7, 1906.



table conditions by sharply striking across the muscle with a ruler or similar instrument, causing a local contraction at the point struck, with a bunching up of the muscular tissue that persists from a few seconds to several minutes.

#### CHAPTER IV.

### TROPIC CONDITIONS.

THE significance of abnormal variations in the nutritional conditions of a part is at once apparent when it is recalled that the growth and nourishment of all the structures of the body are provided over by trophic centers acting through peripheral nerves. For the proper nutrition of skin, muscle, nerve, and bone the integrity of the trophic center, of its peripheral path, and of its termination is essential. In other words, the anterior spinal cell and its polar prolongation in the efferent nerve, the lower motor neuron, can not be injured or destroyed without correspondingly impairing the function of nutrition in its area of distribution. All diseases, therefore, which affect the anterior spinal gray matter or the peripheral nerves are symptomatized by trophic changes in the associated parts. Further, in some local conditions of disturbed nutrition, where even upon minute examination of trophic cells and efferent nerves we can not discern any abnormality with all the means now at command, still we are justified by analogy in supposing some modification of this trophic energy, some dynamic change, some perversion of the function of the trophic apparatus relating especially to its center.

It is also evident that some substances act as stimulants or depressants of the trophic centers. This is seen in the hypertrophies of acromegalia and myxedema, in Addison's disease, and genital adiposity where there is a disordered action of certain ductless glands. Other agents, as perhaps lead, may have a depressing effect upon these centers. The blood-supply of a part and its nutrition are intimately associated. The vasomotor and trophic apparatus are apparently mutually dependent, and as a matter of clinical fact it is constantly observed not only that in wasted tissues the blood-supply is greatly reduced, but that in hypertrophic states the vascularity is increased.

When the trophic apparatus of a limb is involved, the various tissues suffer in proportion as they are highly organized. Fibrous tissue, cuticular epithelium, and bone are but slightly modified, though their growth and repair may be stopped, while the more vascular and highly organized muscles and nerves promptly waste.

**The Skin.**—In conditions of *acute trophic irritation*, as in herpes zoster, the skin becomes injected, red, and painful, and the epidermis is raised in blebs or bullæ filled with serum. When the condition is one of *chronic trophic irritation*, the skin may become thickened, infiltrated, boggy, and show an actual hypertrophy of the subcutaneous areolar structure. The epidermis is increased in thickness, presenting scales and roughness, with an increase in the growth of hair and the secretion of sweat. The nails become rough, striated, coarse, and grow rapidly. *Diminished trophic energy* is marked by opposite conditions. The

epidermis is thinned, imperfect, dry, and non-resisting to ordinary wear; the hair becomes brittle, scant, and sometimes turns gray; the nails grow slowly and imperfectly. The dermal envelope closely binds the underlying parts, loses its pliability and softness, and presents a reddish, purplish, or glistening whitish appearance.

**The muscles** show disturbance of trophic innervation very promptly. Acute poliomyelitis, or division of a peripheral nerve, is followed in a few hours by a muscular relaxation that is quite apparent, and in a few days the part looks wasted, though it may show no change on measurement. At the end of a fortnight a lessened size can be easily demonstrated. The sarcode elements rapidly alter and tend to disappear, finally leaving only the fibrous constituents to represent the muscle by a band of dense tissue, which shortens, gradually producing contractures and deformities.

In those conditions where the process is slower, the muscle does not change in mass, but is gradually invaded by the atrophy and shows some normal fibers very late in the disease and others that may be actually hypertrophied. In pseudohypertrophic paralysis the muscles become infiltrated with fat, which displaces the muscle elements, exaggerates the muscular firmness and outline, and gives a false appearance of strength.

**The peripheral nerves**, when cut off from their trophic centers in the anterior spinal gray matter, show similar degeneration. Under conditions of trophic irritation they present proliferation of their fibrous structures and general thickening of their trunks, with more or less disintegration of their nisher elements. *Ophio-serene atrophy* is a visible instance of trophic impairment.

The loosely organized, almost non-vascular **tendons and ligaments** in adult cases show little change when deprived of their nerve-supply, but even bone itself becomes less resistant, more fragile, and somewhat rarified under these conditions. In children body growth is usually seriously checked.

The trophic condition of **joints** in nervous affections is one of great interest. In tabes dorsalis, and more rarely in other organic diseases of the brain and spinal cord, as in progressive spinal muscular atrophy and cerebral palsy, great changes take place in various joints. They become enlarged, enormously distended with fluid, and the bones disintegrate without any painful attending symptoms. This condition of *dystrophic arthropathy* was first clearly recognized by Charcot, and the articulations thus affected are often called *Charcot joints*. Again, arthritis of any character may be followed by extensive atrophy of the muscles above the joint, principally those of extension. This *arthritic muscular atrophy* in which the muscles waste, but conserve their electrical and reflex responses, is attributed to an irritant condition which arises in the inflamed joint. It affects the trophic spinal centers, and thence acts upon the nutrition of the particular muscles physiologically associated with that joint. It is positively known that division of the afferent path from the inflamed joint to the spinal center prevents the atrophic sequel.

**Decubitus.**—In acute processes involving the spinal cord an acute bed-sore sometimes appears in a few days, and may completely denude



the sacrum. While this is favored by pressure due to the position of the body and moisture from the urinary incontinence that is often present, it may also appear independently. Gross cerebral lesions are similarly marked in some cases by a sloughing of the buttocks, and this decubitus, as in the spinal lesions, is doubtless primarily due to interference with the trophic control, and in the case of hemiplegia affects the paralyzed side only.

**Acute cystitis** in paraplegic states, the low resistance of the tissues to pressure or heat, and the tardiness of repair have a similar significance and add greatly to the difficulty of caring for such patients.

Symmetrical limited deposits of fat and pigment, Raynaud's disease, symmetrical scleroderma, and scleroderma in the form of *straw atrophies* following the nerves are clearly due to trophic disturbances, and are classed among the *trophic aecroses*.

Local conditions of trophic impairment may be a part of general ill-health or systemic diseases, and may in some cases be due to, but should not be confounded with, them. *Doses* will also produce marked nutritional changes in a part, as insensitivity does of the entire organism,—a fact to be borne in mind in relation to neurasthenics, hysterics, and malingers.

## CHAPTER V. ELECTRICAL CONDITIONS.

NORMAL muscles and nerves respond to various methods of electrical stimulation in a precise and uniform manner. On the other hand, diseases which modify the electrical conductivity of nerves or their muscle-endings and diseases which modify the electrical stimulability of muscle-cells produce characteristic changes in their electrical reactions when properly tested. These changes are of very great diagnostic importance. We require, for a reasonable examination of this nature, first, an induced or faradic current that can be increased from zero to a tetanizing strength; and, second, a constant or galvanic current of at least fifty volts' strength, that can also be gradually and uniformly increased or decreased at will or interrupted at pleasure. The first is supplied in many convenient and portable forms; the second, by about thirty active Leclanché or equivalent cells, or the constant incandescent lighting circuit modified by a proper rheostat. For portable purposes batteries of various makers containing from twenty to fifty cells are available. Two electrodes are needed, one presenting from nine to twelve square inches of conducting surface, the other fitted with an interrupting handle by which the operator can easily control the current, and having a rounded conducting surface about one-half inch in diameter.

When using the constant current, a milliamperemeter is of value and for purposes of record almost a necessity. Its readings show the amount of current in circuit at the particular time. It is to be remembered that all alterations in the conditions of the test—such, for instance, as dryness of the electrodes or of the skin of the patient—modify the amount of electricity requisite to secure a given response. Probably identical conditions cannot be secured twice in succession. All ampere-



measurement comparisons are therefore relative and approximate. Cells vary so much in activity at different times that to base the record upon the number used is futile. Where a rheostat is employed in controlling a lighting current of a given potential, an index scale can be arranged that will furnish fairly satisfactory records.

A uniform method of making electric tests is important. The arrangement of electrodes recommended by Erls and found satisfactory in

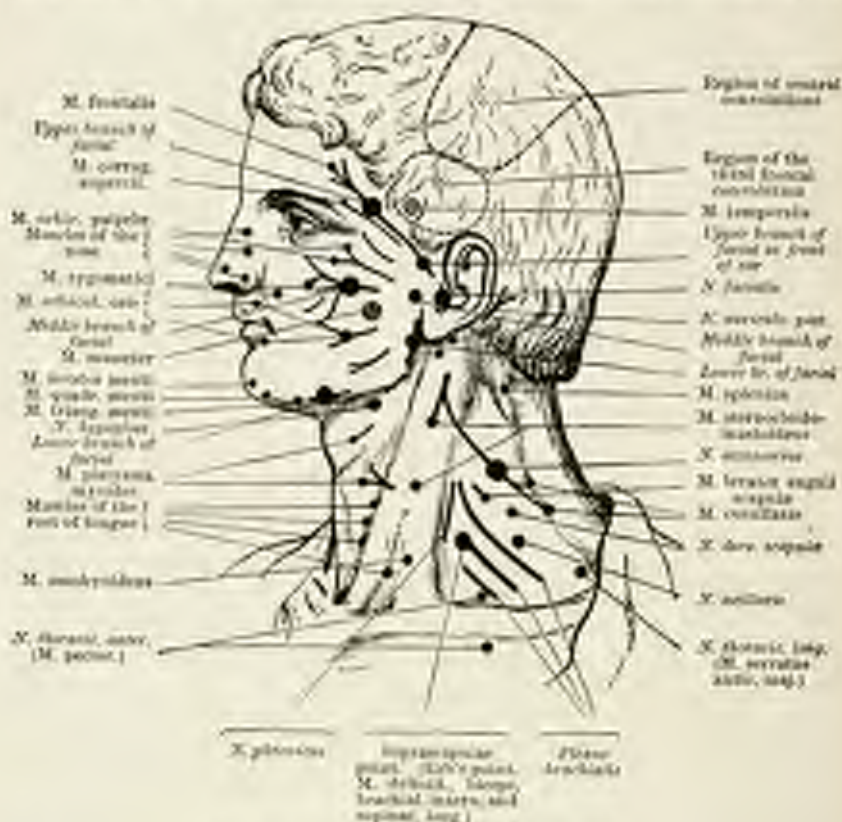


Fig. 6.—Nerves and motor points in face and neck.

all instances is perhaps the best. The large or *indifferent electrode* thoroughly moistened in warm water, with which it is also well to wet the skin, is applied over the sternum, where the absence of muscles and of much sensitiveness obviates disturbing features. The small or *active electrode*, fitted with an interrupter and well moistened, is then placed over the nerve or muscles to be examined, and the current allowed to pass (that is "made") or is interrupted (that is, "broken") as required. This interference, for the sake of brevity, is called the *make and break* of the constant current.

**In Health.**—The nerve-trunk—as, for instance, the ulnar at the elbow—in a healthy person, when stimulated by a mild *galvanic* current, causes all the muscles to which it is distributed to contract more or

less vigorously in proportion to the intensity of the current employed. It is to be particularly noted that upon allowing the current to pass the response is instantaneous, and that the muscular contraction at once reaches its maximum and maintains it until the current is modified. This phenomenon attends stimulation to either the positive or negative faradic pole or electrode, but the negative causes a slightly stronger effect.

If, now, the constant current be substituted, we notice, first of all, that

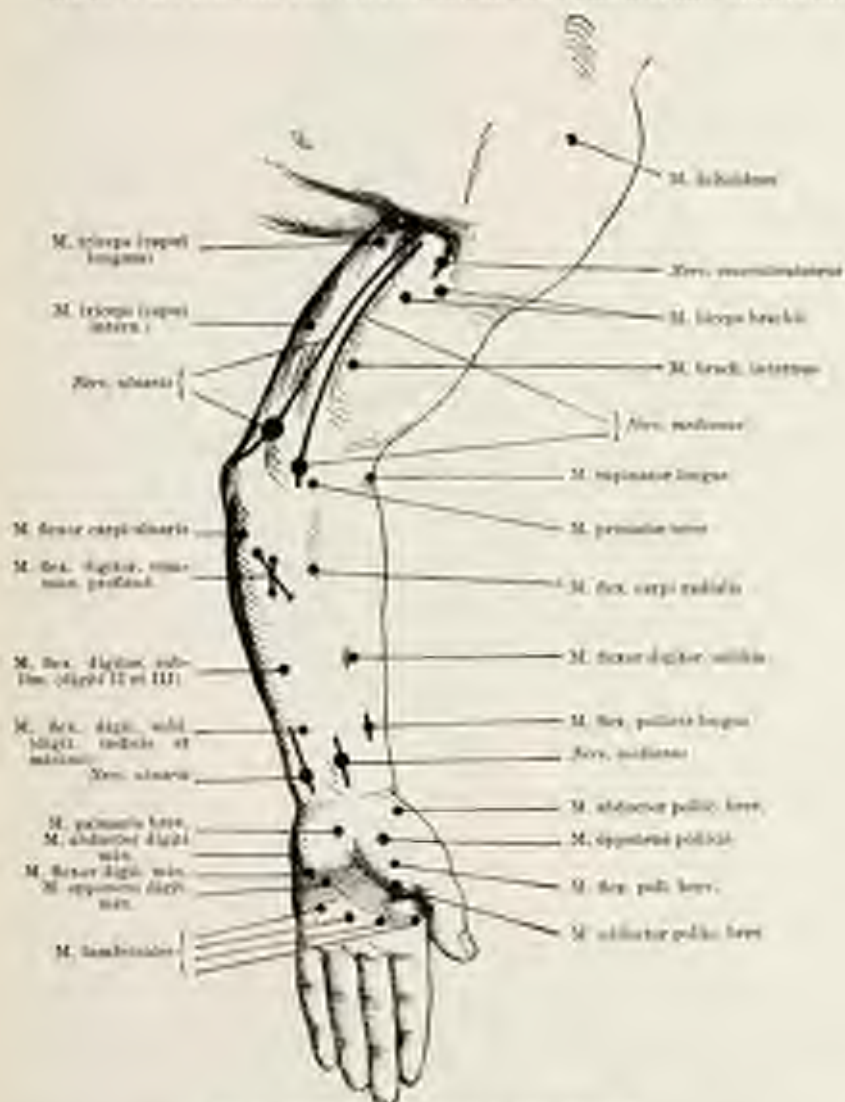


Fig. 3.—Senses and motor points in upper extremity.

mild currents applied to the *nerve-trunk* produce no response. When the current is sufficiently increased it causes, only at the make and

break, single instantaneous contractions of the innervated muscles, which as quickly subside. Looking closer, we notice that the contraction with the negative make—otherwise the cathodal closing contraction (C.C.C.)—is the first to appear as the current is gradually increased, that next comes the positive make, or anodal closing contraction (A.C.C.), followed in turn by the positive break, or anodal opening contraction (A.O.C.), and then, finally, by the negative break, or cathodal opening contraction (C.O.C.). Further, that by the time we have C.O.C., A.O.C.

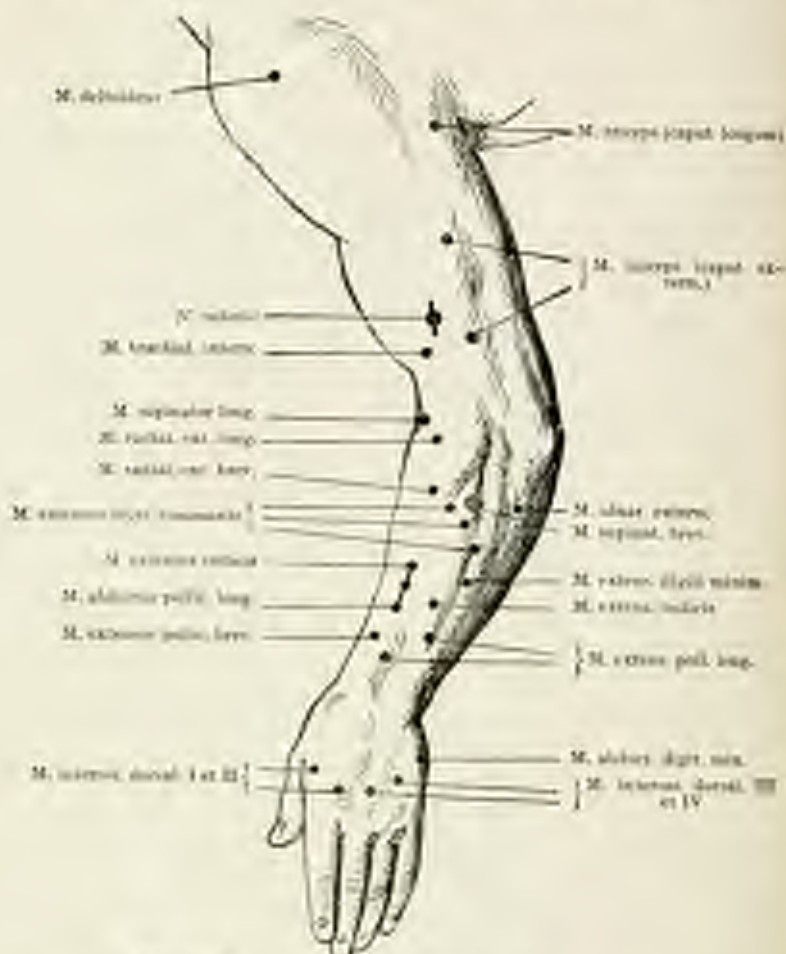


Fig. 18.—Nerve and motor points in upper extremity.

will be vigorous, A.C.C. very strong, and C.C.C. produces a constant tetanic condition lasting as long as the current flows when once it is set up by the make—a condition called cathodal closing tetanus (C.C.T.).

The contractions from stimulating the nerve-trunk thus in health appear in the following order: C.C.C., A.C.C., A.O.C., and C.O.C., and this indicates their intensity in a decreasing order. The



important point to constantly bear in mind is that the negative make contraction through healthy nerves is stronger than the positive. This fact is thus formulated:  $C.C.C. > A.C.C.$

In using the constant or galvanic current one must be sure of the pole with which he is operating. The negative pole can be readily determined by bringing both metal terminals into contact with a piece of wet litmus paper which shows the characteristic acid red color at the spot touched by the negative ead. Or both metal terminals of the conducting cords may be placed in a little water. The decomposition of the water at the negative pole is evident in the formation of numerous fine gas bubbles.

When the active electrode is applied over the belly of a given muscle, a muscular contraction is set up by the *faradic* current, the same as that produced by stimulating the nerve-trunk. If the current be not too strong and the electrode not too large, the effect may be limited to the given muscle, or to a portion of a muscle. Every muscle may also be actuated by the *galvanic* current by applying the active

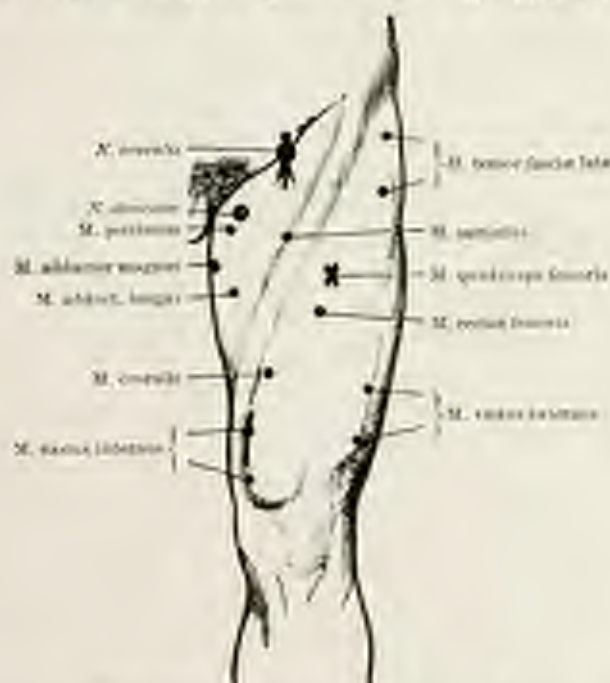


Fig. 11.—Nerves and motor points in lower extremity.

pole on the point corresponding anatomically to the entrance of its motor nerve. A familiarity with motor points is of practical importance. They are shown in the accompanying diagrams. Slight variations in their location are common, and several trials are often required to find them. It is well to mark with a lead-pencil the necessary points, if repeated tests are to be employed. The *galvanic pole responses* are, in healthy muscles, identical with those obtained through the nerve-trunks.

**In Disease.**—Diseases changing the nutrition and structure of the

lower motor neuron, which consists of the spinal cell-body and its peripheral projection and termination in muscular tissue, alter not only the trophic conditions over which the neuron presides, but also interfere with its electric properties. If the spinal cell be destroyed, or its peripheral filament divided, degeneration follows below the lesion, in both the nerve and muscle, with loss of voluntary control. The reactions of such a nerve and muscle to electricity are modified in a highly characteristic

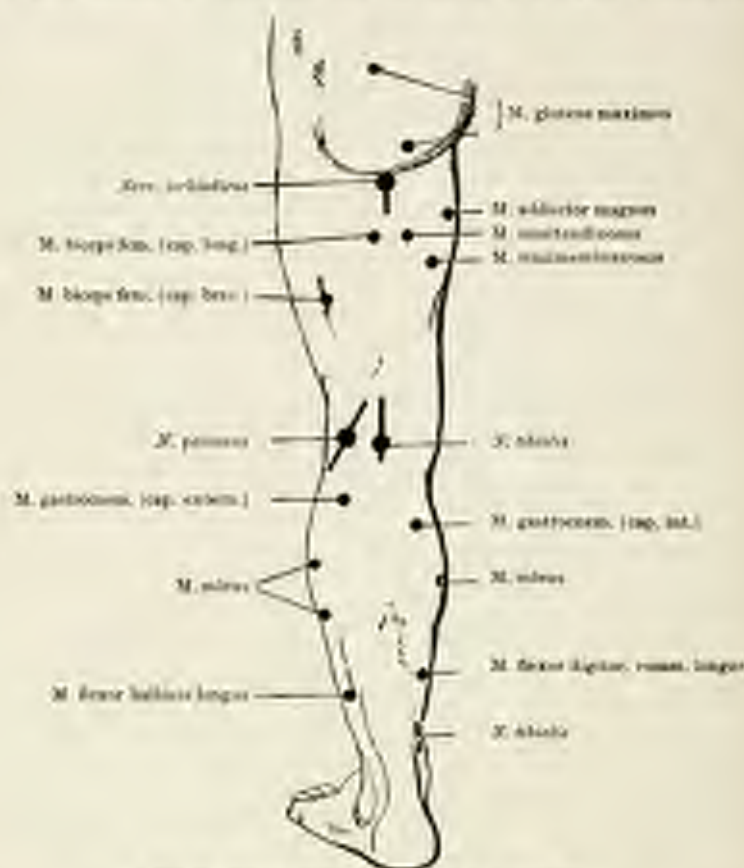


Fig. 12.—Nerves and motor points in lower extremity.

manner. They present the *reaction of degeneration* (R. D.), the presence of which is an indubitable sign of structural change. At the end of about a week, during which there has been a gradual decrease of electric excitability in the muscle and nerve to both faradic and galvanic shocks, four differences become manifest:

- First. The *muscle* responds weakly, sluggishly, and deliberately to faradism, and shows a tendency to maintain the contraction after the current is withdrawn. This is the *nodal change*.
- Second. The *nerve-trunk* loses progressively and equally its responsiveness to both galvanism and faradism—a *quantitative change*.

Third. The muscle becomes much more excitable by galvanism and much less excitable by faradism, which latter reaction with the nerve-trunk responses is completely lost after two or three weeks. This is the *qualitative change*.

Fourth. A *polar change* appears in the muscle about the second week when directly stimulated by galvanism. It is now found not only that the muscle is more readily caused to contract by the constant current, but that the normal mastering strength of the negative closing contraction over the positive has disappeared and that the positive closing contraction is equal to or greater than the negative. Expressed thus: A. C. C. = or > C. C. C. Anodal tetanus (A. T.) is often obtained. This polar difference continues until the nerve either regenerates and regains its normal status, or, failing to recover, gradually the galvanic irritability subsides. It is entirely abolished with all other electrical responses in complete degeneration.

Should recovery take place, the restitution is marked ordinarily, first, by a return of voluntary control, then, by the appearance of galvanic and faradic excitability in the nerve, and, last of all, by faradic excitability of muscle. The hyperexcitability of the muscle to galvanism and

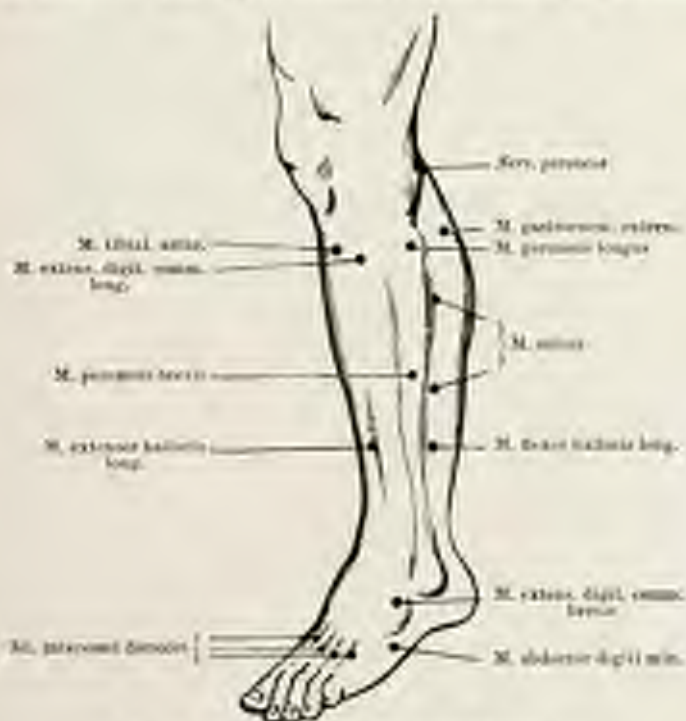


Fig. 13.—Nerves and motor points in lower extremity.

the polar variations from the normal gradually right themselves at the same time. These changes and the pathological process in nerve and muscle are shown in the diagrams (Fig. 14) modified from Erb.

In some cases we find a *partial reaction of degeneration*, the nerves



trunk response being only slightly modified. The inverted polar formula is not necessarily present. The most essential element in the reaction of degeneration is the nodal change,—the slowly appearing muscular response,—so at variance with the instantaneous effect produced in health. Next in importance is the lessened faradic control of the muscle, and, last, the polar modifications. The variations in the galvanic and faradic responses in disease are due principally to the fact that currents of a certain strength and duration are required to actuate the changed sarcoelectric cells, and the rapidly interrupted faradic current thereby becomes inoperative. The galvanic current, if interrupted with sufficient rapidity, is equally powerless.

In some conditions is found a simple increase or decrease of electrical excitability, constituting a pure quantitative change. In those diseases

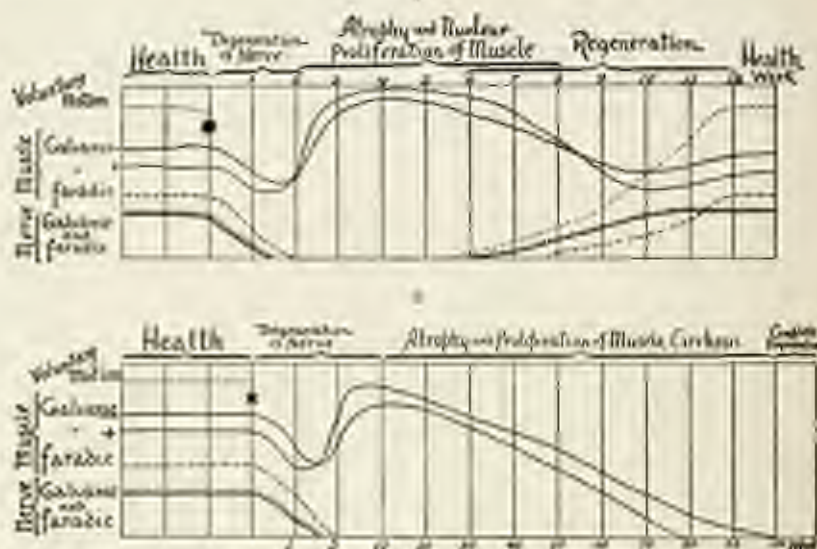


FIG. 14.—1, Paralysis with early source of motion (modified from Kitz). 2, Insensate paralysis with complete atrophy and degeneration (modified from Kitz).

where the muscles seem permanently affected, as in the progressive atrophic myopathies, the responses are reduced, but are proportionate to the amount of healthy muscular fibers remaining. Some spasmodic conditions, like facial tic, present an increased activity to electric stimulation. There is increased electrical excitability in tetany and in Graves' disease. In myotonia we encounter a peculiar electrical response called the *sarcolonic reaction*. In this condition there is normal faradic excitability of nerves, but the electrical excitability of the muscles is greatly enhanced to all forms of current. The positive and negative closing galvanic contractions are nearly equal and all responses are tonic and prolonged. The mechanical excitability of the muscles, as to blows, is similarly exaggerated. In myasthenia gravis the muscular responses are rapidly exhausted by repeated stimulation with faradism or galvanism. They gradually lose force as the stimulus is repeated,

and in a few moments fail completely, but reappear after a short rest. This peculiarity is called the *anæsthetic reaction*.

The use of electricity to test cutaneous sensation is open to so many errors and attended by so much difficulty that simpler means, always at hand, are to be preferred. In those feigning complete cutaneous anæsthesia the surprise of a strong, painful, faradic current from a dry, metal electrode will be likely to unmask the deceit.

When the active electrode is placed over the closed eye or at the temple, the make or break of constant currents is attended by a subjective sensation of light, and this test serves to show the activity of the retina and optic nerve. By placing the indifferent electrode on the same side of the neck, one eye at a time may be examined, and for this purpose only small currents—from two or three cells or of as many milliamperes—should be used. In the same way either *auditory acuity* may be tested by placing the active electrode over the mastoid or before the tragus, the negative pole producing much the louder sound.

For testing the nerves of taste the galvanic current is a prompt and reliable agent. By those of keen perceptions a difference between the positive and negative pole can be distinguished. For the ordinary purpose of taste testing, electrodes ending in probe-pointed metal terminals, or even the metal ends of conducting cords held to the tongue or pharynx, at once give rise to an acid or salty taste, which is quickly appreciated by the patient. One or two cells are all that is required, and one side of the tongue can be easily compared with the other in lateral lesions, or the tongue of a healthy individual will furnish a standard when needed.

For the purpose of localizing the various muscular areas in the motor zone of the cerebral cortex when it is exposed, surgically or otherwise, a mild faradic current is used. It may be applied to the brain surface by means of a wire or pointed metallic electrode, the other pole being attached to a broad sponge placed over the sternum. It is best applied by a double probe electrode, which limits the faradic action to a definite area. Such a current thus applied causes, in the related muscular periphery, similar responses to those produced by faradizing the nerve-trunk, but the movement has more of a purposive or gesticulatory character. These responses also follow an extracranial application of the electrode.

## CHAPTER VI.

### SENSORY CONDITIONS.

MODIFICATIONS of sensation are among the commonest conditions attending nerve-lesions and functional disturbance of the neural apparatus. It is well to have a prefatory understanding of the fact that sensation, of whatever variety or quality, is due to the appreciation of temporary motion and contact, or, in simpler terms, that all sensations are modifications of touch. It is the impact of luminous and sound-waves on properly qualified nerve expansions that gives rise to sensations of sight and hearing. The contact of odorous and sapid particles stimulates the sensations of smell and taste. The recognition of the ca-



rious qualities of objects in contact with the skin gives us information of size, shape, temperature, hardness, smoothness, and a thousand physical properties of the external world.

Another general consideration is that any over-stimulation of sensory nerves is painful. That which is so readily called the pain sense is but the subjective recognition of this overstimulation coupled by instinct and experience with the concept of harm. Thus the contact of a sharp point is at first so recognized and the sense of pain comes when the pressure is increased and the stimulation intensified. It can not be doubted, however, that pain and thermic sensations have courses in the spinal cord separate from tactile impressions. Extremes of heat, cold, pressure, noise, light, odors, and tastes give rise to pain or to discomfort analogous to pain. They cause automatic or conscious efforts of avoidance and self-preservation. It is also a matter of common experience that sensations, except of an extremely painful character, if long maintained, are more or less completely ignored. Therefore, to elicit sensation the motion or contact must be temporary or repeated after distinct intervals. Clothing to which we are habituated gives rise to little or no conscious sensation. With the long persistence of an odor there is a gradually diminishing perception of it. On the other hand, the sudden stopping of a clock or other familiar and disregarded sound is almost startling.

In considering the various qualities and modifications of the sense of touch it is requisite to bear in mind that keenness of sensibility varies greatly not only in different cutaneous areas in a given subject, but considerably in different individuals. The more keen, active, and intelligent the make-up of the subject, the quicker and more precise are the responses. To all tests of sensation the disadvantage pertains that we are dependent upon the responses of the person tested; so that such tests are only partly objective. Sudden extreme pain, however, is usually attended by certain recognizable signs, such as change of countenance, quickened pulse, dilated pupil, pallor, and even perspiration. To carry out the needful tests we require the intelligent cooperation of the patient, and to this end he must understand exactly what is sought. In order that his replies may depend upon his receiving information through the tested source alone, the eyes should be bandaged or other suitable precautions taken to prevent their use. The tested parts should be comfortably placed and supported and all distracting conditions, such as noise or talking, avoided. Check tests must also be used, such as asking "what is felt?" when nothing is applied, or by using some indifferent object in place of the one which the patient is expecting, as the finger-tip instead of the pin point, requiring him at the same time to tell what it is.

The attending conditions must be usual and natural. It is useless to test the sensibility of chilled extremities or to expect reliable replies from the stuporous. In many instances, moreover, there is much sensory disturbance without the patient being aware of it, as in hysteria; or there may be a dissolution of the various qualities of the sense of touch that has not specially attracted his attention, as in syringomyelia.

The **tactile sense** enables us to recognize the contact of objects with the skin. In a crude way its delicacy may be tested by stroking with a feather or flake of cotton, by touching lightly the ends of hairs grow-



ing on the limbs, or by ruler contacts when the sense is found blunted. An instrument called an *esthesiometer*, consisting of two movable points, is frequently employed in testing this element of the sense of touch. A pair of compasses answers the purpose. Observations are made as to the least distances at which two blunted points are both recognized when brought into contact with the skin at the same time, and in a line parallel to the course of the nerve supplied to the part. These distances vary widely, as between the tip of the tongue or finger and the dorsum of the body. The following average measurements will serve as a standard of comparison, or if the sensory disturbance be limited to one side of the body, the opposite corresponding area will furnish a better guide. Differences, to be significant, must be about double those here indicated:

TABLE SHOWING ORDINARY DISTANCES AT WHICH TWO POINTS ARE RECOGNIZED.

Tip of tongue . . . . .	.1 mm. (1-25 in.)	Tip of nose, cheeks, eyelids . . . . .	12 mm.
Tip of fingers . . . . .	.2 "	Temple . . . . .	13 "
Lips . . . . .	2 "	Back of hands . . . . .	20 "
Dorsal surface of fingers . . . . .	4 "	Neck . . . . .	25 "
Tip of nose . . . . .	8 "	Forearm, leg, back of foot . . . . .	40 "
Forearm . . . . .	9 "	Back . . . . .	60-80 "
		Arm and thigh . . . . .	80 "

Another means of testing the tactile sense is to place variously-shaped objects on the skin and to ask a description of them, or to trace letters and numerals with a blunt point. The so-called *stereognostic sense* is a complicated faculty, embracing many or all of the elements of tactile sense. All those perceptions of the qualities of an object received through the sense of touch make up a complex enabling its recognition if repeated with sufficient frequency. Thus we can with closed eyes recognize by touch all familiar objects. In certain brain-states, particularly those involving the sensori-motor cortex, and perhaps in a more limited sense the parietal area, the faculty is lost, constituting a state of *astereognosis* or, better, *stereognosis*.

The *pain sense* may be readily investigated by using a pricking point, but it must not be too fine. A pointed quill or toothpick answers well except in marked *analgesia*. The patient is required to state whether a touch or a painful prick is recognized. If the part examined be hairy, pulling on a few hairs with the fingers or a forceps elicits a sense of pain very effectively.

The *pressure sense*, which is not of great importance, is tested by placing on the skin objects of the same size and external character, such as balls of equal size but variously weighted. A spring instrument fitted with an index and a scale is also used. Where the extremities are thus tested it is necessary to so support them that no notion of the pressure be given by motions communicated to the joints and muscles.

The *thermic sense* can be quickly, though roughly, examined by first breathing on a part with the mouth only an inch away and then blowing upon the same part from a distance of twelve inches. In order to estimate it more exactly it is best to use test-tubes filled with water at various known temperatures. Every test requires that considerable surface come in contact with the skin for a few moments, as the integu-

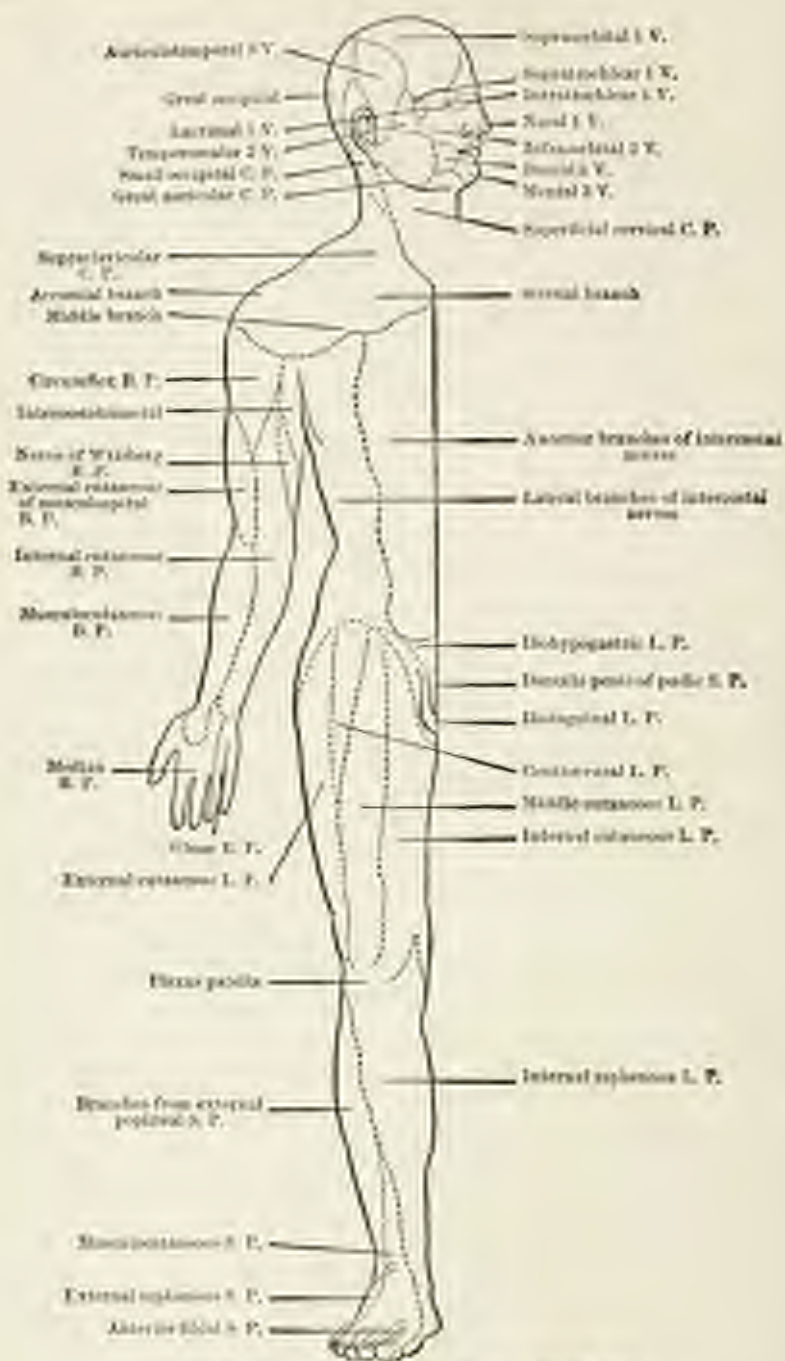


Fig. 41.—Unusually distribution of nerves (after Flower).

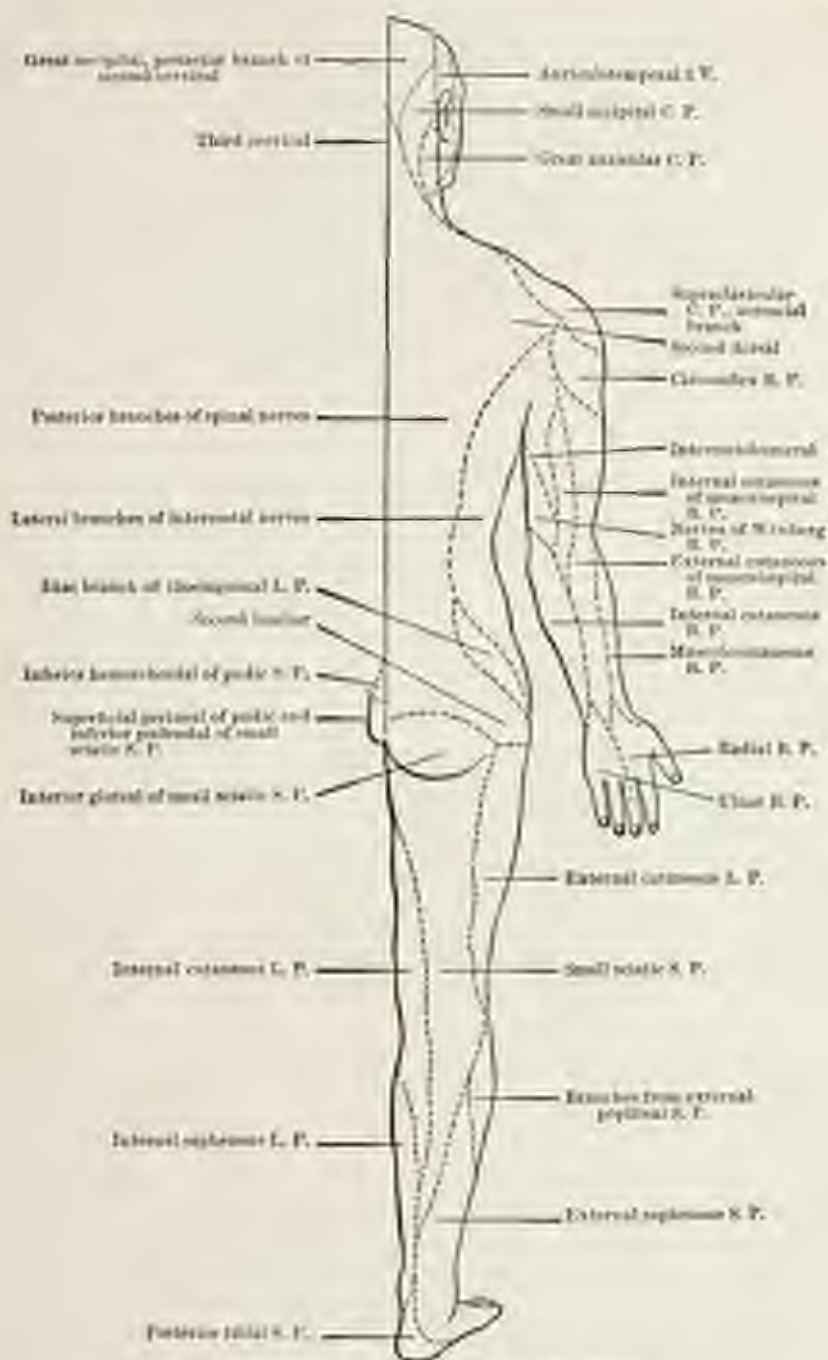


Fig. 26.—Cutaneous distribution of nerves (after Flower).



ment must gain or lose heat proportionately before the exact degree of temperature is appreciated. Within a few degrees of the ordinary body surface-temperature—namely, 80° to 86° F.—slight changes are not recognized in health. Below this to freezing and above it to about 100° F. a variation of two to five degrees is readily perceived. Inability to recognize temperatures from 60° to 70° as cool and 80° to 100° as warm may be considered abnormal. Such a condition is denominated *thermo-anæsthesia*, and, when complete, *thermo-anæsthesia*. Those parts of the skin possessing the most acute tactile sense are also most sensitive to heat.

The term **muscular sense** is used in two ways:

First. It refers to the sensations arising in muscles themselves. In a painful degree this occurs in a cramp or in a tetanizing fascic contraction. The tension on muscles and tendons is no doubt subconsciously recognized by the coördinating mechanism, and it is only when these sensations are greatly intensified that we take conscious note of them.

Second. By muscular sense we refer to the ability to estimate the weight of various bodies. This ability is to so great an extent a result of practice and varies so widely among healthy persons that unless great differences of weight are not appreciated the test gives little information of value. Balls, such as those employed in the pressure test, can be used, or attempts may be made to repeat a certain number on the dynamometer. When the sensation of tendons, joints, and muscles is blunted, not only is the muscular sense reduced, but knowledge of the position of the limbs is wholly or in part wanting and ataxia is present, as described under Errors of Motility.

In certain conditions, notably tabes, *sensation is retarded* and the reaction time is greatly prolonged. A painful impression, as by the prick of a pin on the foot, may not be recognized and responded to for several seconds. In such cases the transmission of all touch sensations may not be equally slow. We prick the patient, having instructed him to say "now" as soon as he feels the needle, and we note the interval, which, in health, is a small fraction of a second only. The intelligence and promptness of the patient materially affect the apparent length of intervening time. Under this test the patient may promptly recognize the contact, but only after a distinct interval of time does he experience the feeling of pain. In some instances a single prick is recognized as two or more, or a painful impression is felt at a distant point,—a *referred sensation*,—or on the opposite side of the body at a symmetrical spot,—*alloesthesia*.

The *sensibility of bones and joints* may be roughly but readily tested by resting the stem of a vibrating tuning-fork at those points where they are subcutaneous. Normally one perceives the communicated thrill.

Complete loss of sensation is properly termed *anæsthesia*. Through usage this word signifies any degree of blunted sensation, and is qualified by adjectives such as partial, complete, or slight, as the case may require, and further limited by such combinations as muscle anæsthesia, tactile, thermic, and joint anæsthesia. The loss of the sense of pain is called *analgesia*, and this word is compounded in a similar way.

A most painstaking study of the varieties and relations of the elements making up general sensibility has been made by Head, who for

the purpose had the cutaneous nerves of his own forearm surgically divided.<sup>1</sup> From his observations it is evident that superficial and deeper sensibilities follow different afferent fibers, and are gathered into a comparatively simple complex higher up. He says: "The sensory mechanism in the peripheral nerves is found to consist of three systems:

"(I) **Deep sensibility**, capable of answering to pressure and the movement of parts, and even capable of producing pain under the influence of excessive pressure or when the joint is injured. The fibers subserving this form of sensation run mainly with the motor nerves, and are not destroyed by division of all the sensory nerves of the skin.

"(II) **Protopathic sensibility**, capable of responding to painful cutaneous stimuli, and to the extremes of heat and cold. This is the great reflex system, producing a rapid, widely diffused response, unaccompanied by any definite appreciation of the locality of the spot stimulated.

"(III) **Epicritic sensibility**, by which we gain the power of cutaneous localization, of the discrimination of two points, of very light touch, and of the finer grades of temperature, called cool and warm."

Sensation may be intensified, giving rise to *hyperesthesia* and *hyperalgesia*. These conditions are made evident by the usual tests, and require no extended review.

In addition there are a host of purely *subjective sensory disturbances*, described as sensations of heat or cold, numbness, prickling, crawling, creeping, tingling, heaviness, deadness, etc.,—*paresthesia*. Areas so affected may show no alteration of sensibility when actually tested. Paresthesia usually are symptomatic of general nutritional states or of the so-called neuroses.

Having determined a localized *dysesthesia*, or condition of disturbed sensation, it is of the first diagnostic importance to outline it as accurately as possible. Sensation may be disturbed by lesions which involve sensory paths at any point from the cerebral cortex to the terminal organs in the muscles and skin. The *dysesthetic area*, however, presents a different and distinctive outline as various nerve-levels are injured.

If a nerve-trunk or branch be injured by traumatism or disease, anesthesia will be limited to the corresponding anatomical cutaneous distribution. *Per contra*, if such anatomical area be found to be anesthetic, the inference is at once justified that a peripheral lesion is present. As soon as the sensory nerve-fibers reach the spinal cord they ramify so widely in the various segments that it would be impossible for any spinal disease or injury to select a given number from all others. Figures 15 and 16 show these sensory areas, and should be carefully studied.

If the *spinal cord* undergo a complete cross-lesion or division, we have loss of sensation in the nerve-area below the upper level of the injury. Now, the distribution of anesthesia has relation to the cord-segments, and not to the nerve-trunks. For a practical understanding of this fact it is necessary to recall that the body, from one end of the vertebral chain to the other, is made up of a number of similar segments or links, all more or less perfectly represented in the skeleton, muscles, viscera, skin, and nervous apparatus. In the dorsal region the plan is comparatively simple, but as the limbs are tracked it is greatly

<sup>1</sup> "Beiss," 1905 and 1908.









sharp, lancing, or dull and heavy? Note exactly the *distribution* of the pain. Observe if it is limited to the cutaneous area of a peripheral nerve or if it has the distribution of a spinal segment. In every instance of pain, seek for *tenderness*. The painful part, during the presence of protracted pain, is almost invariably sensitive. When lightly touched, certain portions of the hyperæsthetic surface will be found especially affected. These *maximal points of pain* are usually situated where the sensory nerves become superficial or pass bony prominences.

It is best to carefully outline these sensitive areas with a blunt point, as the head of a large pin, and not trust to the rough gestures and statements of patients. In other instances the tenderness can only be detected by deep pressure. For instance, intercostal neuralgia produces

superficial tenderness; pleurisy is marked by tenderness on deep pressure or percussion. To Dana and Head<sup>1</sup> we owe a definite knowledge of the relation of cutaneous sensitiveness and pain to visceral disease. Head's work, abundantly verified, clearly shows that the superficial surface of the body is in relation with the viscera through the spinal centers. Disease arising in the visceral portion of a vertebral segment gives rise to pain referred to the body or somatic area. Similarly, disease of the spinal segments gives rise to pain and other sensory disturbances in the skin, as already pointed out, and in the associated viscera. Mackenzie<sup>2</sup> insists that the viscera are insensitive and that pain is felt through the cerebrospinal nerves alone. That, for instance, the



Fig. 18.—Application of the scheme to the trunk (Bismuth). Aneurysm is indicated by horizontal lines; hyperæsthesia by diagonal lines.

well-known tenderness over the liver readily elicited by pressure in disease of that organ is in fact confined to the skin and muscles of the abdominal wall. These areas, as mapped out by Head, with their maximal points for the body and limbs, are presented in figure 20 (A, B, C), and the following table shows the relation of the viscera to these areas:

TABLE SHOWING AREAS OF PAIN REFERRED FROM VISCERAL DISEASE.

- Heart*.—First, 2d, 3d, dorsal segments.  
*Lungs*.—First, 2d, 3d, 4th, 5th dorsal.  
*Stomach*.—Sixth, 7th, 8th, 9th dorsal; cardiac end from 6th and 7th.  
     Pyloric end from 9th.  
*Intestines*.—(A) Down to upper part of rectum.  
     Ninth, 10th, 11th, and 12th dorsal.  
     (B) Rectum.  
     Second, 3d, and 4th sacral.

<sup>1</sup> Head, "Brain," 1st.

<sup>2</sup> "Brain," 1902.

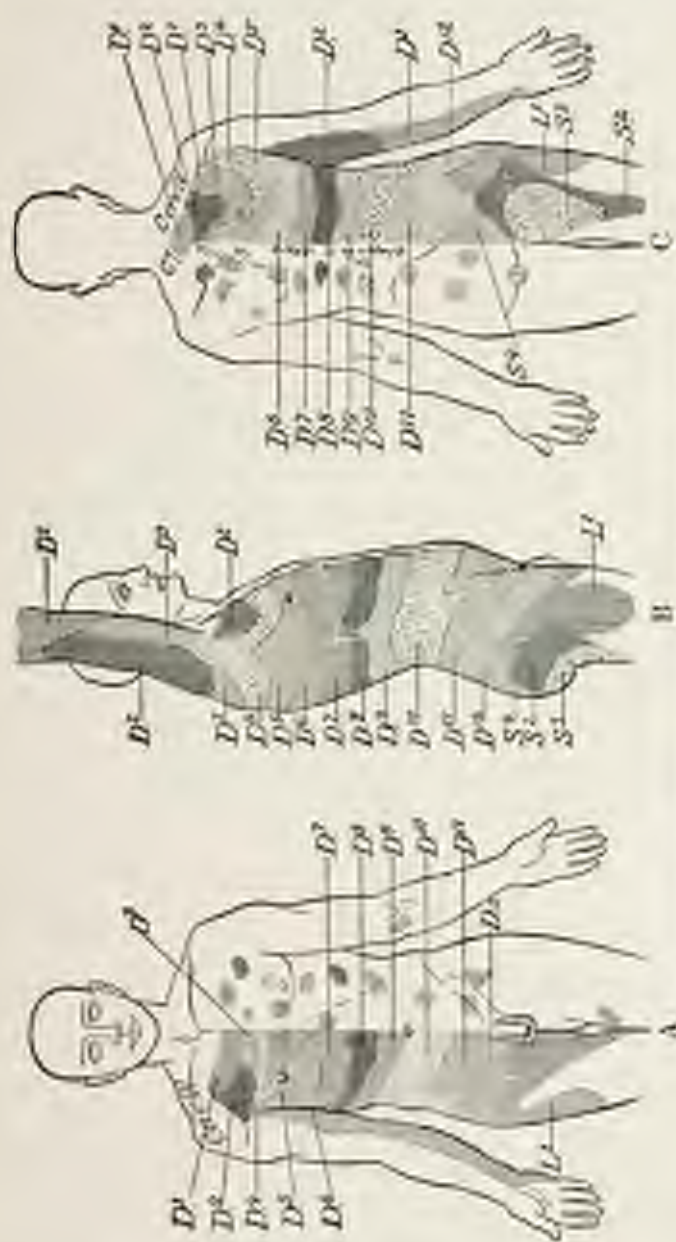


Fig. 26.—Cutaneous areas and maximal points of pain related to the vertebral segments (after Brack).



*Liver and Gall-bladder.*—Seventh, 9th, 9th, 10th dorsal.

Perhaps 5th dorsal.

*Kidney and Ureter.*—Tenth, 11th, and 12th. The source the lesion lies is the kidney, the cause is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter, the more does the 1st lumbar tend to appear.

*Bladder.*—(A) Mucous membrane and neck of bladder.

First, 2d, 3d, 4th sacral.

(B) Overdistention and ineffectual contraction.

Eleventh and 12th dorsal and 1st lumbar.

*Prostate.*—Tenth, 11th, 12th dorsal.

First, 2d, 3d sacral and 5th lumbar.

*Epididymis.*—Eleventh and 12th dorsal and 1st lumbar.

*Testis.*—Tenth dorsal.

*Ovary.*—Tenth dorsal.

*Appendages, etc.*—Eleventh and 12th dorsal and 1st lumbar.

*Uterus.*—(A) In contraction.

Tenth, 11th, 12th dorsal and 1st lumbar.

(B) Os uteri.

First, 2d, 3d, 4th sacral and 5th lumbar very rarely.

A further point elucidated by the same author is the relation of head pains and aches to visceral disturbances. Such pains and aches are marked by definite areas of sensitiveness when at their height and by ten-



Fig. 18.

Fig. 19.



Fig. 20.

Figs. 18, 19, and 20.—Maximal points of referred and associated pain in the head and neck.

der maxima. These maxima, as those upon the trunk, are practically the tender points of Vullix. They are much more persistent than the tender areas, which usually are only well marked when the pain is severe and gradually subside concentrically, leaving the maxima. On the head and face, as on the trunk and limbs, these pain-areas have segmental distribution and in no manner correspond to the peripheral nerve-supply.

Figures 21, 22, and 23 show the cephalic maximal points, and the following table shows the relation of the body-viscera to these head-points and also to the body-areas:

TABLE SHOWING ASSOCIATED PAINFUL AREAS ON THE HEAD RELATED TO VISCERAL DISEASE IN THE BODY.

Attack on Viscer.	Associated Area on Head.	Organ in Painful Relation to These Areas.
Cervical, 3 . . . . .	Frontonasal (7 notal).	Apices of lungs. Stomach. Liver.
Cervical, 4 . . . . .	Frontonasal.	
Dorsal, 3 . . . . .	Midorbital.	Lungs. Heart. Ascending arch of aorta.
Dorsal, 3 . . . . .	Midorbital.	Lungs. Arch of aorta.
Dorsal, 4 . . . . .	Dorsalital.	Lungs.
Dorsal, 5 . . . . .	Frontotemporal.	Lungs. Heart.
Dorsal, 6 . . . . .	Frontotemporal.	Lower lobe of lungs. Heart.
Dorsal, 7 . . . . .	Temporal.	Basal of lungs. Heart. Stomach.
Dorsal, 8 . . . . .	Vertical.	Stomach. Liver. Upper part of small intestine.
Dorsal, 9 . . . . .	Parietal.	Stomach. Upper part of small intestine.
Dorsal, 10 . . . . .	Occipital.	Liver. Intestine. Ovaries. Testes.
Dorsal, 11 . . . . .	Occipital.	Intestine. Fallopian tubes. Uterus. Bladder.
Dorsal, 12 . . . . .	Occipital.	Intestine. Uterus, etc.

In like manner disease within the head and neck has its referred pain, associated tender area, and maximal point. They may be thus tabulated:

Organ at Fault.	Maxima on Face and Throat.	Organ at Fault.	Maxima on Face and Throat.
Chiasm (neuritis) (cause of accommodation).	Mid-orbital.	Lower first and second molars.	Hyoid and pain in the ear.
Cornea.	Frontonasal.		
Iris . . . . .	Frontotemporal.	Lower third molar.	Superior laryngeal.
	Temporal and maxillary.	Mandibular (temporal).	Hyoid.
Vitreous (glaucoma).	Temporal.	Middle ear.	Vertical and behind ear.
Retina.	Vertical.		
Teeth (upper incisors).	Frontonasal.	Tongue, tip.	Mental.
Upper canine and first bicuspid.		Tongue, lateral part.	In ear and hyoid.
Upper second bicuspid.	Nasalal.	Tongue, base . . . . .	Superior laryngeal.
Upper second bicuspid.	Temporal or maxillary.		Occipital.
Upper first molar.	Maxillary.	Tonsil.	In ear and hyoid.
Upper second and third molars.		Nose, olfactory portion.	Frontonasal and nasalital.
Lower incisors, canines, and first bicuspid.	Mandibular.	Respiratory portion and posterior nares.	Nasalal.
Lower second bicuspid.	Mental.	Larynx.	Superior and inferior laryngeal.
Lower second bicuspid.	Mental or hyoid.		

*Brain disease* presents pain of two varieties: First. When the meninges are involved there is a local pain with tenderness on pressure and percussion. Second. In conditions of intracranial pressure and disease of the brain proper there is superficial tenderness and the pain is widely distributed over the head. In the second-class of cases pain is usual in the brow, vertex, occiput, back of the neck and shoulder, and similarly distributed tenderness is common.

The descriptions of some pains by patients are classical and almost diagnostic. The lightning pains of tabes, the gnawing pains of rheumatism, the burning pains of neuritis, the girdle pains of spinal disease, the lead-cap pressure pain of acromasthenia, the pain under the breast and in the groin in hysteria, the daily recurring brow pain of malaria, and the nocturnal pains of syphilis have a significance quite their own. The circumstances under which pains occur often throw light upon their nature. Sciatica and lumbago are provoked by motion and allayed by rest. The head pains of eye-strain bear a distinct relation to ocular employment. Neurasthenic pains always increase under fatigue or depression—that is, exhausting—emotions. A pain that is practically circumscribed has a tendency to spread to associated organs and to neighboring areas after long duration, general depression, or the onset of any marked physical illness. It thus becomes generalized.

## CHAPTER VII. THE SPECIAL SENSES.

**Sight.**—The eye presents many interesting and valuable symptoms in a wide variety of nervous affections. Its systematic examination should be a part of the case-taking in every instance.

The lids on the two sides may show a difference in the palpebral opening due to paralysis or spasm. Exact symmetry is the rare exception in health, but any marked acquired inequality, unless due to scars or local conditions, such as conjunctivitis, irritation, swelling, new growths, etc., implies some variation of muscular control or nerve-supply. A falling of the upper lid, or *ptosis*, is a common early symptom of tabes and syphilitic brain disease, while in a facial palsy an inability to close the lids is a marked sign. From the same cause the lower lid may be everted. An acquired prominence of the eyeball may greatly enlarge the palpebral opening. In exophthalmic goiter the lids frequently fail to follow the upward and downward excursion of the ocular globe. This is not entirely due to the protrusion of the eye, as it has been noted in the absence of this condition, and in some instances is congenital. In hysteria a condition often confounded with ptosis, but really an orbicular spasm, is sometimes encountered. *Blepharospasm* as a limited facial tic is a very common affection. Many states of brain and optic-nerve disturbance are marked by the closed lids of *photophobia*, which should not be confounded with that arising from inflammatory conditions of the lids, cornea, or iris.

Attention has already been directed to the reactions and reflexes of the pupils. In all examinations of the pupils the observer must ex-



clude the actions of those drugs, like opium, cocaine, and belladonna, which modify their size and reactions. Irregularities in their outline or inequalities in size are to be carefully noted, but pupillary deformities from antecedent inflammatory processes or injuries followed by synechia must not be mistaken for perverted innervation. Inflammation of the pleura,<sup>1</sup> apical tuberculosis, and pneumonia may cause either narrowing or widening of the corresponding pupil when tested by ordinary daylight. Paralysis of the pupillary muscles is *iridoplegia*; paralysis of the ciliary muscles—*cycloplegia*—is marked by the loss of the function of visual accommodation. They are usually found together, and then constitute *ophthalmoplegia interna*. Paralysis of the external muscles of the eye—namely, the recti, obliqui, pathetici, and the elevator of the lid—is denominated *ophthalmoplegia externa*.

The external muscles receiving their innervation from three sources, the third, fourth, and sixth cranial nerves, are very frequently involved singly or in groups. This gives rise to various deviations of the visual axes of the eyes or loss of power in directing them conjointly in some given direction, with resulting indistinctness of vision or complete double vision,—*diplopia*. Rarely a *monocular diplopia* is encountered as a pure hysterical symptom, but it may be the result of defective curvatures in the ocular media, as in corneal deformities. The special examination to determine the muscle or muscles at fault in these squint conditions will be taken up under the consideration of the diseases of the cranial nerves distributed to the ocular apparatus. Great and unwarranted stress has been put by some enthusiasts upon a condition of a lack of balance among the extrinsic muscles of the eye, named *heterophoria*. Of much greater importance are errors of refraction and accommodation in myopia, hyperopia, and astigmatism. They are attended by conscious or unconscious efforts at clear vision, constituting a condition of *eye-strain* that may furnish an active source of nerve waste. Extreme and constant deviations from the normal control of the extrinsic eye muscles can, no doubt, act in the same way, but slight variations in conditions of ill-health are commonly the result and not the cause of such states. As the general state fluctuates, they correspondingly vary for better or worse.

Vision can be readily tested by the types of Jaeger, and when seriously defective, by having the patient count fingers held in a good light against a dark background. Astigmatic error is roughly and quickly shown by the use of the numerous familiar charts for the purpose. For further details reference should be had to systematic works on the eye.

The ophthalmoscope is one of the most important instruments in the diagnostic outfit of the neurologist. An ability to readily examine the ocular fundus at the bedside or elsewhere is one of his most needful accomplishments. Familiarity with the normal ophthalmoscopic picture, supplemented by experience in recognizing vascular disturbances, choked disc, and atrophy of the optic nerve, will often render positive a host of otherwise obscure indications. The characteristic appearance of the fundus in diabetes and Bright's disease and the choroidal changes of syphilis are valuable signs.

<sup>1</sup> Chassagnard, "Arch. Gén. de Méd.," Mar., 1885.

The *visual field* is the area over which objects are visible while the eye is fixed. In health its limits are tolerably uniform, being modified above and within by the brow and nose. In this field colors of objects are recognized by the normal eye at various distances and in a certain order from the *fixal point* upon which the gaze is directed. From without inward come white, blue, yellow, orange, red, green, and violet.

The form of the test-object is perceived before its color is apparent, as is shown by the chart (Fig. 24).

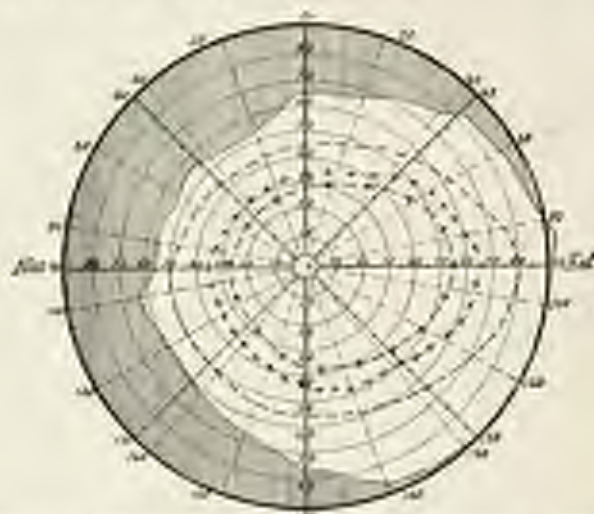


Fig. 24.—Normal visual field for form (and the various colors) (after Neisser). Form field indicated by the line field is indicated thus ————, the red field thus + + + + +, the green field thus ○ ○ ○ ○ ○.

The visual field is peculiarly modified by various diseases. In pronounced *hysteria* we have usually a concentric uniform reduction of the field. The color-fields may be reduced almost to the fixing-point or entirely obliterated. Even more characteristic is a rearrangement of them so that the red field overlaps or completely surrounds the blue. The relation of red and blue is therefore to be remembered. Cushing and others have found contraction, inversion, and interweaving of the visual color fields rather common in *brain tumor* cases, and not apparently dependent upon the extent or duration of the usually attendant *lobes*. In *severe* cases the fields are frequently much reduced, and fatigue conditions promptly increase their contraction. In well-marked cases the effects put forth by the patient in responding to the tests may serve to greatly increase the reduction of the fields within a few minutes. In *alcohol* and *alcohol poisoning* and other toxic conditions the fields are sometimes greatly contracted and present irregular blind areas, or *scotomata*. Destructive diseases occurring lack of the globe may cut off a portion of the field, producing *hemianopia*, *scotomata*, *central blindness*, *eccentric blindness*, or blindness in a quadrant of the field, as the fibers or centers related to the given area are involved.

To test the field of vision a perimeter is of service, and, for accurate examinations and records, indispensable. Roughly, it can be done by



placing the patient opposite a fixed point on a bare black or dark wall, at a fixed distance of eight to twelve inches. With one eye covered he is ordered to maintain his gaze unswervingly on the fixed point. A small white object, preferably about a centimeter in diameter, is brought into the field from the periphery along various lines radiating from the fixed point, and the spot marked at which it is first perceived by the patient. By joining a series of such points the outline of the field is constructed and the various color limitations are similarly defined by noting the distances at which the color of the test-object is clearly recognized. Large blind spots may in this way sometimes be detected, the test-object being carried across the field to the fixing-point. The normal blind spot corresponding to the optic papilla must not be mistaken for a symptom of disease. More roughly still the field can be rapidly tested by facing the patient at a distance of about two feet. He is then directed to look you steadily in the eye opposite the one to be tested; that is, if the patient's left eye is under examination, he looks at the examiner's right. A small object is brought into the field of view in a plane midway between the patient and physician, and the distance at which it is seen is noted. At the same time the examiner's own perception furnishes a check and measure to the test.

The subjects of *Daltonism*, or *achromatopsia*, are congenitally defective in color perceptions. Some have complete *achromatopsia*, everything to them being pechably of a neutral tint; others do not distinguish some elementary colors, as red from green; and others, again, fail to detect marked shades of the same color. This defect would modify tests of color-fields accordingly.

**Hearing.**—The sense of hearing is most often modified by local conditions in the meatus and middle ear. Our usual problem is to determine whether the nerve-apparatus is impaired. To this end we note at what distance on either side the patient can hear a watch, remembering that in advanced years such high-pitched sounds are not heard so readily as lower tones, like those of the voice. If the watch is not heard through the air, the ear should be closed by pressing in the tragus with the finger and the watch brought into contact with the root of the zygoma, the mastoid process, the parietal bone, or the teeth. If it is now heard, the difficulty is presumably in the conduction apparatus, and not in the nerve. This can be confirmed by using a tuning-fork in Rinne's test. Set it in vibration and place the handle against the mastoid or zygoma, the meatus being closed. When no longer heard, unstop the ear and hold the still vibrating fork close to it. The normal ear will detect tones through the air that do not reach it by bone-conduction, but if there be obstruction in the external or middle ear, the bone-path will be the more acute. The formula  $B. C. > A. C.$  or  $B. C. = A. C.$  If there is no recognition of high or low tones by bone-conduction, the nerve-apparatus is undoubtedly diseased, or if with hearing greatly reduced  $A. C. > B. C.$  is still found, the probability is that the nerve is diseased.

*Auditory hyperesthesia* is occasionally encountered in acute cerebral meningeal conditions and in hysteria. Severe headaches, meningitis, and



many cerebral affections are marked by *dynorexia*,—sounds producing discomfort,—which may or may not be attended by real auditory hyperæsthesia. In the relaxation of the tensor tympani muscle attending facial palsy low notes are heard with unusual distinctness, while those of high pitch may not be so clearly perceived as in health.

Subjective sounds, varying from an insignificant tinnitus to pronounced and formulated auditory hallucinations or loud explosions in the head, are referred to with great frequency by nervous invalids. Their starting-point is not rarely in the external or middle ear or Eustachian tube. Irritation of the auditory centers and nerve, however, is sometimes the cause.

Involvement of the labyrinth or of its nerves is marked usually by vertigo, and in extreme cases by forced movements in a given direction, which, as in Ménière's disease, may furnish attacks of great suddenness, throwing the patient to the ground. The *quære sensu* with equilibrium is disturbed. (See also p. 134.)

Finally, in no case of obscure brain symptoms should the possibility of extension of infection from suppuration in the middle ear and mastoid be forgotten,—a condition that specular examination and local searching commonly reveal.

**Smell.**—The sense of smell may be reduced or obliterated in one or both nostrils. It is necessary to test them separately, closing firmly the opposite anterior nares. The inspiratory efforts should not be too vigorous, as thereby the test odor may reach both nasal spaces through the pharynx. In selecting the test-material, pungent odors or irritants, such as ammonia or tobacco-smuff, should not be employed. In hysterical anæsthesia of the mucosa may be associated with absence of the sense of smell, so that the strongest irritation gives rise to no response; otherwise, when the olfactory nerve is completely destroyed, stimulants and irritants have their usual effect. It is also well to choose an odor with which the patient is familiar, and to recollect that people vary greatly in keenness of scent. The sense of smell is greatly impaired by nasal-catharrhal trouble, and is often practically lost during a severe cold in the head. Degeneration of the fifth nerve, which supplies common sensation to the nasal mucous membrane, also lessens its acuteness.

Occasionally the sense of smell is greatly intensified. Hallucinations of smell are rare, and in several cases have been found to depend upon disease in the temporosphenoidal lobe.

**Taste.**—Pure taste sensations are the recognition of bitter, sweet, sour, and salt. When a taste is associated with an odor, we speak of a *flavor*, and it requires the participation of the olfactory nerve, usually stimulated by way of the posterior nares. Flavors, in consequence, are lost with the loss of smell, and not with the loss of taste. The margin and tip of the tongue are more sensitive to sour and salts, while the base and pharyngeal pillars best recognize bitter and sweet. The entire gustatory area, which includes the dorsum of the epiglottis and even a portion of the rima glottidis, as well as much of the pharyngeal wall, distinguishes all tastes more or less readily.

For the purpose of testing taste, solutions of sugar, quinia, citric acid, and salt, or the powdered substances, answer. The tongue should be

protruded and the test-substance applied to a small area. Some moments are usually required before the taste is perceived. The galvanic electric current furnishes a simple and reliable agent. Two probe-pointed metallic electrodes separated by a few millimeters are placed on the portion of the tongue to be tested, and a non-painful current from one or two cells, of a few milliamperes' volume, is used. A metallic taste is elicited.

Taste is lost from the tip of the tongue in lesions of the facial nerve involving the chorda tympani. Hemiplegic states rarely show a one-sided loss of taste, while the hemianesthesia of hysteria, when pronounced, is usually marked by apathy on the same side.

Perversions of taste—*parosmia*—and increased sensitiveness—*hyperosmia*—are sometimes encountered in neurotics and hysterics. Subjective taste sensations are also rare, but may furnish the aura of an epileptic attack in the so-called uncinata fits, or be dependent upon local irritation of the trunks of the nerves of taste, as in ear disease affecting the chorda and facial nerve.

## CHAPTER VIII.

### SPEECH.

THERE are as many kinds of speech as there are avenues to consciousness and routes therefrom. We have spoken language, written and printed speech, gestures and emotional attitudes that portray thought and serve as media for the communication of ideas. Consequently, speech may be modified by disease in innumerable ways as the successive levels of the nervous system are invaded, and every variety of speech may be disturbed either in its perceptive or emissive channel.

The emission of vocal speech requires mechanically the coordination of the mouth parts, the larynx, and the chest-muscles of respiration. *Musculariations* of the nose, throat, mouth, and larynx are attended with difficulty in shaping the resonant chamber for the precise modifica-

PHYSIOLOGICAL TABLE OF CONSONANTS.

	VOICELESS ORAL CONSONANTS.	VOICED ORAL CONSONANTS.	VOICE NASAL CONSONANTS.
Labials . . . . .	P	B W	M
Labiodentals . . . . .	F	V	
Lingvodentals . . . . .	Th S	Th Z	
Anterior lingualpalatals . . . . .	Sh T	Zh D L R	N
Posterior lingualpalatals . . . . .	K H or Ch	G Y	Ng



tions of vocal sounds in the production of spoken language. Cleft-palate, closed nasal spaces, and tied-tongue are not uncommon conditions of this variety. They cause difficulty mainly in the pronunciation of the consonant sounds. If we are familiar with the physiology and mechanism of consonant production, we have the key to diagnosis.

For this purpose the preceding chart, slightly modified from Wyllie,<sup>1</sup> is of great importance. He divides consonants into voices less and vocal, as their pronunciation is or is not devoid of laryngeal sound. By having the patient pronounce these letters, or phrases containing them, the seat of the difficulty is at once recognized. For instance, in the palatal pulse of diphtheria the posterior linguopalatals and the nasal resonants, especially Ng, which depend on the separation of the nasal spaces from the pharynx by the soft palate, are slurred or lost.

**Stammering, or stuttering,** is a speech defect attended by difficulty in attacking properly the enunciation of words beginning with certain consonants. These consonants are reiterated with more or less spasmodic respiratory effort, and finally the word either drops out or is explosively spoken. There is always more or less incoordination in the needed muscular combinations.

Speech in idiots and infants often consists of the iteration of a syllable or single sound (*babbling*) or the repetition of the final word or phrase overheard by them, without reference to its meaning,—*echolalia*. In multiple sclerosis the speech becomes deliberate and each syllable is pronounced with the distinctness of counting. In general paresis the words are jumbled. The patient catches in the middle of words, repeats syllables, slurs sounds, and omits terminals or even important words. This is called *syllable doubling*. In *hereditary ataxia* the speech shows the incoordinate control of the musculature of vocalization and is usually slow, monotonous, and unmodulated. In cerebral palsies with athetosis speech may be characteristically modified by the spasmodic actions of the muscles of the tongue, throat, and chest. It is explosive, spattering, now slow, now fast, and the tone qualities are uncontrolled. In *hysteria* persistent *aphonia* is a frequent symptom, a whispering voice only remaining. Complete *mutism* may also develop slowly or suddenly in this malady, but voice sounds, as in coughing or sneezing, usually remain to show the acoustic character of the disturbance. In *progressive bulbar palsy* the paralysis of the tongue is early marked by indistinctness of speech and a loss of the lingual consonant sounds. If the lips are weakened, the labials can not be produced, and, finally, through paralysis of the tongue and larynx, vocal speech is reduced to inarticulate noises.

In diseases marked by tremor, as in *alcoholism*, *Graves' disease*, and *paralytic syphilis*, the voice is also tremulous. Depression, excitement, and the emotions generally are manifest in the timber and modulation of the voice. The deaf are inclined to speak in a monotonous, high, or more often low, tone that is quite peculiar to them.

**Aphasia.**—Organic disease of the brain, throwing out of operation the various cortical centers related to speech or breaking up their connecting channels, produces peculiarly interesting phenomena that require

<sup>1</sup> "Disorders of Speech," Edinburgh, 1891.



very careful examination. Any of the qualities or varieties of speech may be affected, or almost any combination of defects may be present in a given case. Practically we have to investigate both spoken and written speech and to determine: first *how* they are received and apprehended, and, second, *how* conceived and expressed. In other words, we try to determine whether the difficulty lies in the entrance-channel or the receptive center on the one hand, or in the formulating center and the emissive route on the other.

Take, first, the *reception of spoken words*. Is the hearing good, tested by watch, tuning-fork, voice, and various sounds? If so, does the patient understand the words used; or is he *word-deaf*—that is, while hearing words does he fail to appreciate their meaning? Test this by directing him to execute certain movements—to shut his eyes, clasp his hands, etc.

Second, *how* does he *produce spoken words*? Is he reduced to a single expletive or phrase, or is he completely dumb? Does he forget names of common objects (*anomia verbata*), stammer, slur, stumble, or reiterate? Does he miscall objects with which he is familiar (*paraphrasia*), and is he aware of his mistakes? Is his speech a jargon of meaningless sounds or words strung together like beads? If dumb, can he write his answers? and if he can not write, can he indicate with his fingers the number of syllables in the names of objects pointed out to him? Finally, can he repeat or echo what is said to him, or is he inclined to echo his own words or expressions? does he understand his repeated words?

**Written Speech.**—If vision is good, does the patient understand written or printed questions? This can best be determined by writing simple commands, as, stand up! sit down! give me your hand! and not by questions that can be answered by a nod. Proper responses show comprehension. If the written questions or commands are only partially understood, we must attempt, by repeated tests, to decipher the limitations of his *ofusia*. Secondly, does he write? If *agraphia* is not present, does he use wrong nouns (*paraphrasie*), repeat letters or words, or make serious omissions? Can he write from dictation and from copy, and does he then understand what he thus writes?

When other speech avenues of the mind are blocked or only partly distracted, the recognition of *gestures*, their use and repetition, should be noted. Some patients do not make gestures (*anomia*), or employ wrong gestures in attempting to explain themselves (*paranomia*).

To some patients objects have lost their meaning, so that familiar things and intimate friends are not recognized,—a condition called *word-blindness*. The sense of touch (*stereognosis*) may still furnish information to the mind that has lost its recognition of visual impressions, so that a piece of money or familiar object unrecognized by sight may be correctly named when placed in the hand, though this faculty is also commonly defective when there is *word-blindness*.

**The handwriting**, especially with the pen, in many cases furnishes diagnostic evidence of great value, as well as a means of studying the progress of the disease. A hand-magnifier will often bring out peculiar-

arities that are not readily seen by unaided vision, and for the same purpose photographic enlargement may be used. When abnormalities are slight or only suspected, a specimen from something written several months or years previously will serve as a proper basis for comparison. Appropriate allowances for youthful growth or the decrepitude of age must be made. The education, habitude of writing, and physical condition at the time are also to be considered. As a rule, in health the down strokes are made with more strength, precision, and rapidity than the other written lines. If they show inequalities, tremor, waves, or marked angularities, the significance is greater than the appearance of these anomalies in the upward lines or connecting curves. The signing of the patient's name, for those who write it frequently, becomes quite automatic, and often fails to fairly show the character of the disturbance. It is well to dictate some ordinary sentence, to have the patient copy a paragraph from a newspaper, or write a little account of his illness. Many times it is only after writing a few minutes that the difficulty manifests itself. This is particularly true in writers' cramp. In general paresis the first of a letter page may be well, firmly, and coherently written, the latter part showing tremor, inequalities, omitted words, and incoherence. Blobs, spotters, and wavering lines demonstrate the staxia of the patient. The aphasic shows his cerebral lesion by using wrong words, by writing jargon, and by the repetition of letters, syllables, and words or phrases when not intended.

The loss of complex motor activities, such as agraphia, motor aphasia, amimia, or professional or skilful motor combinations, is denominated apraxia, and may be present without attending paralysis of the corresponding muscular organs.

*Micro-writing* is a variety in which the letters are formed backward, like printers' type, and appear properly when viewed in a reflecting surface. Left-handed children often write thus naturally, and it has been noticed in hysterics and degenerates. Rare cases of *micro-speech* have been recorded, in which words were inverted by syllables or literally.

Varieties of handwriting are given in the description of the various diseases which present such peculiarities.

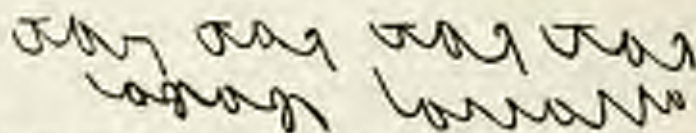


FIG. 264.—Micro-writing spontaneously produced by a five-year-old left-handed child.

Finally, *photography* furnishes a most valuable adjunct to case-taking when any peculiarity of conformation, attitude, gait, or facial expression is observed. Serial photographs vividly present the course of the disease. The use of a *case-book*, properly prepared, is of the utmost value to systematize the examination and secure a full but concise record. The form given on page 71 may serve as an outline, requiring to be properly spaced for actual use.

## FORM FOR NEUROLOGICAL CASE-BOOK PAGE

Name \_\_\_\_\_ Nationality \_\_\_\_\_ Date \_\_\_\_\_  
 Residence \_\_\_\_\_ Age \_\_\_\_\_ Civil Condition \_\_\_\_\_ Children \_\_\_\_\_  
 Occupation \_\_\_\_\_ Heredity \_\_\_\_\_  
 Health History \_\_\_\_\_  
 Injuries \_\_\_\_\_  
 Habits \_\_\_\_\_  
 Present Illness \_\_\_\_\_

## PRESENT CONDITION.

Alimentary System	Eyes
Circulatory System	Lids
Respiratory System	Pupils
Tricardiac System	Movements
Gastro-intestinal System	Vision
Pulse Temperature Weight	Field
General Appearance	Fields
Altitude	
Gold	
	Ears
Motor Condition	Deformities
	Ringing
	Sense of Smell
Splinters	Mouth
	Lips
Sensory Conditions	Tongue
	Palate
Subjective	Articulation
Objective	Voice
Muscular	Aphasia
Integument	Dysphagia
Thermic	Taste
Diminished	Brain
Intensified	Memory
Pain	Logical Powers
	Emotions
Electric Conditions	Characteristics
	Vertigo
	Sleep
Trophic Conditions	Spinal Cord
	Superficial Reflexes
	Deep Reflexes
	Coordination
Diagnosis	Spinal Column
Treatment	Handwriting



## PART II.

# DISEASES OF THE CEREBRAL MENINGES AND CRANIAL NERVES.

### CHAPTER I.

#### THE CEREBRAL MENINGES—PACHYMEINGITIS AND PIAL HEMORRHAGE.

**Anatomical Considerations.**—The coverings of the brain are admirably suited to protect it from injury and infection. Guarded externally by the skull and the scalp-pail, it is intimately enveloped by the dense, fibrous *dura mater* in a practically sealed sac. The usual anatomical descriptions of the cerebral meninges are misleading. Ordinarily three distinct membranes are named and described, when in reality there are but two. Lining the skull we have the *dura mater*, serving as an internal periosteum for the cranial bones and furnishing in part their vascular supply. It is entirely free from the brain, but gives off sheaths to the cranial nerves and the large vessels at their exit from the skull, and supplies venous channels or sinuses for the return circulation of the encephalon. The dual fold between the cerebral hemispheres and the tentorium cerebelli afford support and protection.

In normal conditions the brain fills the cranial cavity fully, and its soft covering membrane is everywhere in contact with the inner surface of the *dura*. The interval which separates them is called the *subdural space*. No actual space, however, exists, the two membranes being smoothly applied to each other and only separated by disease or mechanical means.

Closely investing the brain is the *pia mater*, made up of two layers or membranes very loosely attached by delicate meshes of fibrous tissue. The outer can be easily stripped from the under layer, and constitutes what is usually described as the arachnoid. The alleged double layers and spaces of the so-called arachnoid can not be demonstrated and do not exist. This outer pial layer we may call the *arachnoid pia*. Between it and the under layer, or *visceral pia*, is a varying space, the *pial space*, filled with a delicate, open, reticular network of fibrous tissue containing cerebrospinal fluid. At the gyral grooves the *visceral pia* dips to the bottom of the sulci. It everywhere closely adheres to the brain-cortex, which it follows through the transverse fissure into the ventricular chambers, furnishing the *velum interpositum* and bearing the choroid

pia mater. The *arachnoidea* bridges over the sulci. This arrangement at the great fissures and at the base of the brain in the intervals between the cerebrum, cerebellum, and medulla forms cerebrospinal fluid reservoirs, which are continuous with the pial spaces of the spinal cord through the foramen magnum. Delicate processes of the pia also accompany the cranial nerves and vessels from the skull, and are continuous with the extracranial lymph-channels.

Between the pial layers the cerebral vessels ramify. As they penetrate the cortex both arteries and veins are accompanied by delicate



FIG. 15.—Diagram of cerebral meninges and cortex. D., dura mater; S.D., subdural space; P.S., pial space; A.P., arachnoidea; V.P., vascular pia; A., arterial perivascular space; V., venous perivascular space (Tuke).

sheaths of the visceral pia, which form the perivascular spaces of the brain. Some of the great pyramidal cells of the cortex are encased by diverticula from these perivascular channels, and are thus nourished (Tuke). In a manner, therefore, these most important cortical elements may be considered as appendages of the pia, with which they have such intimate anatomical relations. The pia also pushes up the Pacchionian bodies into the vascular area of the dura near the venous sinuses at the vertex. These bodies are supposed to furnish an outlet for the meningeal fluid.

The brain is constantly changing in bulk through variations in vascularity due to mental or physical activity. This would be impossible



were it not for the ready displacement of the cerebral fluid. Losses of brain-substance by atrophy or otherwise are mechanically compensated in bulk by an increase in the pial fluid and the hydrostatic balance is preserved. It is probable that the entire pial surface participates in the production and absorption of the cerebrospinal fluid to some slight extent, but the choroid plexuses or choroid glands, as Frazier prefers to call them, are the chief producers. The wide ramifications of the pial structure through the substance of the brain, its lymphatic prolongations, and its ventricular relations make plain many of the symptoms of meningitis and account for some of the serious sequels of the disease.

#### INFLAMMATION OF THE DURA MATER—PACHYMEINGITIS.

The dura mater, though a tough, resisting, fibrous structure, is subject to inflammatory invasion. As the outer or inner surface is affected, we speak of *pachymeningitis externa* and *pachymeningitis interna*.

**Pachymeningitis externa** is not a clinical unit. When the outer surface of the dura becomes inflamed, it is always a secondary condition, the result of the extension to it of infection from adjoining structures. Fractures of the cranium attended by sepsis, osteitis, necrosis, and new growths in the cranial bones may be its starting-point. Suppuration of the middle ear sometimes propagates inflammation to the dural covering of the temporal bone. A cranial gumma may incite it. Usually it is limited in extent. Very rarely considerable accumulations of purulent material between the dura and the skull may strip the membrane from the bone and occasion cerebral disturbance by localized pressure.

When inflamed the dura becomes thickened and strong adhesions to the inner surface of the cranial bones are formed. The danger consists in a resulting sinus-thrombosis or in septicæmia. The treatment is that of the surgical condition of which the pachymeningitis is the sequel.

**Pachymeningitis interna**, *pachymeningitis hemorrhagica*, or *hemorrhage of the dura mater*, is a chronic inflammation of the inner surface of the dura mater marked by one or more hemorrhagic membranous layers.

**Etiology.**—The arterial changes resulting from alcoholism are frequently the cause of this condition. In undoubted instances the congestion following alcoholic abuse has determined the vascular rupture that furnishes the laminated membranes. In dementias, and especially in parietic dementias, it is a common post-mortem finding. Infectious maladies, the exanthemata, erysipelas, and carbuncle states, especially those marked by purpuric conditions, as scarlet fever, sometimes lead to it. It is more frequent among men than among women, and appears, as a rule, early or late in life.

**Pathological Anatomy.**—Internal pachymeningitis is essentially characterized by the thickening of the dura and the deposition internally of laminated new membranes of hemorrhagic origin.<sup>1</sup> Raswedsen<sup>2</sup> insists that the first change is proliferation of the epithelial layer

<sup>1</sup> Meyer, "Path. Report, III. Eastern Hosp. for Insane," 1886.

<sup>2</sup> "Ziegler's Beitrag," Bd. xxvii.



followed by fibrinous exudation and the formation of thin-walled capillaries and that the primary condition is a toxemia. Barnett<sup>1</sup> thinks that intravascular separation of fibrin is a constant initial feature followed by the other changes and finds such resulting membranes free from bacteria. These layers vary in number from two or three to as many as twenty, and in consistency from that of freshly extravasated blood to tough, well-organized, leathery membranes. They present, according to their age, the colors of blood under similar circumstances elsewhere. The new ones are bright red, the older ones yellow. They are only slightly adherent to the dura and to one another by fibrous connections, and are quite vascular when of some age. Their blood-vessels are delicate, poorly developed, and readily degenerate, thus furnishing new hemorrhages, which separate the older layers or form new ones on the cerebral surface. Adhesions to the arachnoid are practically wanting. The cerebral convolutions are flattened if the stratified new formation attains considerable proportions, and the cranial bones may also present pressure atrophy. In children the ossification of sutures and fontanels is retarded. This form of dural disease is usually found at the vertex, in the distribution of the middle meningeal arteries over the motor zone, but occasionally the basal fossæ are involved.

**Symptoms.**—The symptoms of the early stage before hemorrhages have occurred, and when the process is purely inflammatory, are very vague. In many cases no suspicion of the disease has been raised during life. The first recognizable symptoms attend the formation of a hematoma large enough to produce cerebral indications. These consist of pain in the head, intellectual troubles, loss of memory, awkwardness in muscular movements, insomnia, vertigo, rarely vomiting, limited or Jacksonian convulsions, apoplectic attacks, rigidities and monoplegias presenting remissions. The temperature is febrile and unconstructive. During the convulsive attacks it attains a high degree, but in the intervals may be subnormal, normal, or slightly elevated.

**Course.**—As the early symptoms escape recognition, the duration of the disease is indeterminate. It usually runs a protracted course, and may in rare instances terminate in recovery, with resorption of most of the new tissue. As it is practically an expression of a serious or hopeless underlying condition, the ordinary end is death. This may



Fig. 26. Pachymeningitis interna in a middle-aged man. The different layers, with cotton-wool plaques (removed), are seen on the right side of the skull internally (Sunderland).

<sup>1</sup> *Bull.*, 1902.

follow an apoplecticiform seizure or a comatose condition, but usually is preceded at intervals by a number of such attacks, each of which leaves a certain trace behind it in the form of added mental or motor disability.

**Diagnosis.**—The diagnosis is difficult. In drunkards, demented, and epileptic children the appearance of the cerebral symptoms mentioned should call the disease to mind. A history of preceding apoplecticiform attacks, with practically complete remissions, would strengthen the suspicion. The thickened membranes form anatomically a subdural tumor, and in times present all the symptoms of a new growth in that locality. The osseous cystitis of syphilitic meningeal involvement and other local features will usually differentiate that disease. The distinction from tubercular meningitis in children rests upon the absence of emaciation, abdominal retraction, severe headache, rigidity of the neck, and intense respiratory and circulatory troubles. In adults, cerebral apoplexy usually is of more rapid onset, but cerebral thrombotic softening often presents a parallel symptom group.

**Treatment** consists of measures to relieve the basic condition. Alcoholic addiction, infectious diseases, and the exsiccated having received appropriate attention, the various cerebral indications are met as they arise. Quiet, the ice-cap, elevation of the head, anti-spasmodics, cathartics, diaphoresis, hot foot-bath, and other means to decongest the cerebral circulation will be of service during the convulsive attacks. Ergot should not be advised, as the bleeding vessels, devoid of muscular layers, would be placed at a still further disadvantage by the increased arterial tension. Morro and Ballard reported a number of cases in adult alcoholics successfully treated by trephining and the evacuation of the clots. When the diagnosis can be made, such procedure is urgently indicated.

#### PIAL HEMORRHAGE.

**Pial hemorrhage**, or *meningeal hemorrhage*, takes place either outside of the arachnoid, on the inner surface of the dura, or within the meshes of the pæa, or in both locations at once. The *retrospinal* variety of meningeal hemorrhage may be found at all ages, but is most common in the new-born. In about one-half of the cases still-born children present this accident, apparently due to protracted labor, sometimes to instrumental delivery, and even to precipitate birth. In one-third of the cases of asphyxiated new-born, Cruveilhier claims that subdural hemorrhage is the main difficulty. The clots are commonly found over the convexity, and are, in surviving cases, a pregnant source of idiosy and cerebral palsy.

Later in life pial hemorrhage is usually produced only by extreme violence to the skull, as in concussion injuries or fractures. The hemorrhage may come from a dural sinus or from the meningeal arteries. It practically requires a fracture with displacement or a penetrating wound to cause sinus bleeding. The location of the middle meningeal artery in a bony channel at the anterior inferior angle of the parietal



bone, a frequent seat of fractures and direct violence, renders it particularly liable to injury. In many cases the hemorrhage from the meningeal vessels takes place at the centre comp. point. Certain debilitating and infectious conditions predispose to and may rarely cause subdural hemorrhage; for instance, hemophilia, purpura, small-pox, scarlatina, typhus, typhoid, and acute rheumatism. It is found in chronic alcoholists, especially after a detoxic. A ruptured meningeal aneurysm may flood the subdural space.

The onset is acute and marked by apoplectic seizure and rapidly developing unconsciousness. In traumatic cases the patient not seldom rallies more or less from the first bewilderment of the concussion, and after a variable interval of minutes, or even hours, sinks into unconsciousness. The cortical irritation is manifest in one-sided or more circumscribed and repeated convulsions, rigidities, and tremors. There is usually retinal congestion and obscuration of the optic disk. The spinal fluid is generally uniformly tinted or deeply colored with blood. The pupils dilate unevenly, the coma becomes profound, the pressure indications intensified, and the patient usually dies in from twenty-four to seventy-two hours, unless relieved by operation.

*Subacute hemorrhage* takes place within the pial spaces, or strips the pia from the cerebral cortex. It is the usual meningeal hemorrhage of adult life, and its common location is toward or in the basal region.

The extravasated blood may widely infiltrate the pial spaces or merely form diffuse ecchymotic discolorations. In large quantities it may force itself into the ventricles through the transverse fissure, and even travel down into the pial spaces of the cord. Usually it forms a thin covering over the surface of the convolutions, dipping into and filling the interconvex sulci. Under the hemorrhage the surface of the cortex often appears softened and lacerated. Ordinarily the blood is of arterial origin; rarely it comes from veins and very exceptionally from a ruptured sinus. Except in traumatic cases, disease of the vessels, such as aneurysm, siliary aneurysm, sclerosis, atheroma, fatty degeneration, or acute infections softening of their walls, furnishes the essential element of causation. It follows that periarteritis and endarteritis are the initial steps in the process that eventuates in hemorrhage. The development of these conditions is taken up more at length under the diseases of cerebral arteries in Part III, to which the reader is referred.

The onset of this form of meningeal hemorrhage is also acute and apoplectic, with a rapid downward tendency toward death. There may be partial temporary restoration to consciousness, but the patient presents indications of great shock and feebleness. A recurrence of apoplectic symptoms is usually quickly followed by death. Previous indications of localized disease, such as aneurysm of the basilar or other large vessel of the base, may have been present in the form of cranial-nerve palsies with the usual features of endocranial tumors. The apoplectic seizure is usually less sudden and violent than in cerebral hemorrhage of the ordinary capsular variety, and often rather gradually develops the full apoplectic state and profound coma. Hemiplegia and conjugate deviation of the head and eyes have been noted in rare cases, but their absence is the rule, and aids in differentiating this condition from cerebral hemorrhage. Epileptiform and tetanic features are



occasionally present and imply cortical irritation. The pulse may be slow and respiratory difficulty present. At first the rectal temperature is reduced, but returns to the normal and rapidly mounts as death approaches. This takes place, as a rule, in from a few hours to a few days. Rare cases have endured a week, and still rarer ones survived.

The diagnosis of intrapial hemorrhage is difficult. It closely resembles cerebral hemorrhage, cerebral thrombosis, and the hemorrhage arising from pachymeningitis interna. The chief symptoms on which reliance is to be placed are the rapidity of onset and the quickly increasing symptoms without paralysis and convulsions. An internal pachymeningitis usually presents a significant history of headaches, and occurs in a limited class of patients with gross degenerations. The differential features of cerebral hemorrhage and softening are tabulated in Part III.

The outlook is extremely grave and death is almost certain to terminate the case. In these days of aseptic cranial surgery an exploratory opening is allowable, and furnishes practically the sole means of controlling the hemorrhage and saving the patient.

## CHAPTER II.

### INFLAMMATION OF THE PIA MATER.

**Leptomeningitis, acute cerebral meningitis, cerebrospinal meningitis, purulent meningitis, inflammation of the soft cerebral coverings,** is an acute, sometimes epidemic disease of a hetero-infectious character, consisting of inflammation of the pia mater and marked by an irregular clinical course.

A general description of leptomeningitis will be followed by an outline of some of its more important clinical varieties.

**Etiology.**—The pial structures can be invaded in but two ways: by traumatic or destructive lesions of the bony and fibrous envelopes of the brain on the one hand, or through the vascular avenues on the other. To the first group belong those cases of meningitis arising from direct inoculation, as in cranial fracture and septic extension from neighboring foci in the scalp, face, cranial bones, the middle ear, the mastoid cells, the nasal fossæ, antra, and sinuses, and from the orbit and pharynx. To the second group belong the larger number, which, formerly called *idiopathic*, are now known to depend on microbial infection. The exact infection path is often problematical. Ortman pointed out the presence of coryza in a large proportion of the cases, and supposed that the infection reached the pial space through the lymph-channels of the nasal vault, which are continuous with those of the brain.

In the epidemic form Scherer<sup>1</sup> again calls attention to the severe nasal catarrh at the commencement of the attack. Weigerts in eighteen cases found purulent nasal catarrh, and demonstrated Weichselbaum's diplococcus intracellularis meningitidis in the secretions. In fifty per-

<sup>1</sup>"*Consigli e Ricerche*," April, 1893.

sons not suffering from the disease *he* found this diplococcus in two instances and supposes that it is inhaled, taken up by the leukocytes, and by them carried into the lymph-spaces of the brain through the nasal vault. Further importance has been given to this pathway by the examinations of Westenhöffer.<sup>1</sup> Flexner and Barker<sup>2</sup> emphasized the probability of the infection atrium being in the intestinal tract, but more recently Flexner has subscribed to nasal invasion as the most common in the epidemic variety. This has been very fully sustained by others, and especially by Dehec.<sup>3</sup> Herrick, Medlar, and others<sup>4</sup> have demonstrated the diplococci in the blood-stream repeatedly in very early stages of the disease even antecedent to brain symptoms; later they tend to disappear from the blood. The hematogenous infection origin of the disease is, therefore, quite probable. Flügge estimates that under epidemic conditions the number of carriers is ten to twenty times that of meningitic cases. Droplets from the respiratory tract are supposed to convey the infection. The close relation of pneumonia to meningitis has for a long time pointed to the lungs as an invasion route. As a clinical fact, any infection at any near or remote point may induce meningeal inflammation by way of the vascular system.

Barterologically, a case of acute meningitis may present one or many organisms. Those most frequently found alone are Weichsbaum's diplococcus, Koch's bacillus, the pneumococcus, the streptococcus, the typhoid bacillus,<sup>5</sup> and the bacillus coli. In association with them, and perhaps often as a secondary infection, are found the staphylococcus aureus and albus and various indeterminate streptococci and bacilli. The most common of all and the most significant are the bacilli of tuberculosis, the pneumococcus, and the diplococcus intracellulæris. These microorganisms are found in the meningeal fluid, but may, as in a general pneumococcal infection, be widely distributed throughout the body. Lately the diplococcus of Weichsbaum has been demonstrated in the pus of arthritis occurring in a case of meningitis, and also in the blood.<sup>6</sup> Osler, in the Cavendish lectures for 1899, made the following practical classification:

ACUTE MENINGITIS.				
ACUTE MENINGITIS.	Primary	1. Of serotropical type.	(a) Specific. (b) Epidemic.	Diplococcus intracellulæris.
		2. Pneumococcal.	Germs alone or mixed, or in a general pneumococcal infection.	Pneumococcus.
	Secondary	1. Tuberculous.	(1) Secondary to pneumonia, endophthal- mia, etc.	Bacillus tuberculosæ.
		2. Pneumonic.	(2) Secondary to disease of lungs or extension to the brain.	Pneumococcus.
		3. Pyogenic.	(a) Following local disease of cranium or a local infection elsewhere.	Various forms of the pyogenic and strep- tococci.
		4. Miscellaneous acute infections.	(b) Transient infection in various chronic diseases.	Typhoid bacillus, tubercu- losis bacillus, diplo- theria bacillus, pneumo- coccus, etc.

It will have been noticed that leptomenigitis is found in frequent association with the other infectious diseases. Curschmann has noted it in small-pox and scarlatina, both with and without purulent

<sup>1</sup> "Berlin Klin. Wochs.," June 12, 1903.

<sup>2</sup> "Ann. Jour. Med. Sci.," Feb., 1894.

<sup>3</sup> "Recherches Épidémiologiques sur la Méningite," Paris, 1901.

<sup>4</sup> "Jour. Amer. Med. Assoc.," June 26, 1918, p. 227; *Ibid.*, Feb. 16, 1918, p. 418.

<sup>5</sup> Daddi, "La Sperimentale," July, 1884. Ohlacher, "Jour. Amer. Med. Assoc.," Aug. 28, 1897.

<sup>6</sup> Gynn, "Phil. Med. Jour.," vol. 5, No. 24.



otitis. It is not rare after typhoid. It is common with pneumonia. All pyemias, whatever their source, have their recorded cases. Acute articular rheumatism is frequently attended by meningeal symptoms which are customarily attributed to rheumatic inflammation of the serous brain-envelope, but Ball,<sup>1</sup> in at least 3 out of 69 such cases, found a purulent effusion, and serous exudation was present in 33. Mumps are often associated with meningitis and epidemics of both have been concurrently noted. The same is true of coryza, which, like parotitis, is sometimes undoubtedly due to the bacillate micrococci. A most marked relationship exists between pneumonia, influenza, and meningitis. They may follow one another in a given patient. They are apparently interchangeable in a gripped epidemic, and present remarkable bacterial analogies. In pneumonia of the apex a meningitic disturbance sometimes arises that is not marked anatomically by any evidence of inflammation. Cultures in such cases have also been negative.<sup>2</sup> The clinical picture, however, is that of acute meningitis, and further investigation may yield positive findings. The so-called acute serous meningitis is generally secondary to some pyemic condition, and in some instances the clear meningeal fluid has contained streptococci.<sup>3</sup> Isolation is undoubtedly at times attended by a meningeal congestion that may develop into active inflammation with a tendency to chronic changes.

**Pathological Anatomy.**—The pathological changes in the meninges are more or less circumscribed when due to infection by extension, and are then often limited to the neighborhood of the primary lesion. On the other hand, infection by way of the circulation gives rise to a generalized meningitis which may be most intense at the vertex or the basal region. The dura mater, except at the site of bone disease or similar infection center, is practically intact and is readily removed. The pia presents a ragged, mottled appearance. The vessels are engorged with blood and the pial spaces are filled with a serous, milky, or purulent exudate, which follows the vascular courses, fills the sulci, and, if sufficiently abundant, unbrokenly covers the convolutions. At other times the exudate appears in discrete patches, which are, for the most part, found at the basal outlets of the cranial nerves and vessels. Extensions sometimes accompany the auditory nerve into the internal meatus or follow the optic nerve into the orbit. The seropurulent deposit is more or less fibrinous, and the serous exudate sometimes is filled with flaky masses, which escape with it when the skull is opened. Ordinarily the inflammation follows the choroid plexuses into the ventricles, which are oftentimes dilated by the increased turbid, flocculent, pial fluid.

The cerebral cortex, in cases of short duration and in the so-called serous form, may show little more than the evidence of increased vascularity. In severe and protracted cases minute hemorrhages are common, both in the pial spaces and the brain-substance. The perivascular sheaths are blocked with exudate, and the cortex is edematous, infil-

<sup>1</sup> "Thèse de Paris," 1893.

<sup>2</sup> Bergé, Calise, "Traité de Médecine," vol. vi, p. 529, Paris, 1901.

<sup>3</sup> Kolesom-Gard Delastre, "Annales de Méd. et de Chir. Infantiles," April 13, 1900.



trated with pus, and adherent to the pia, which cannot be separated from it without stripping off the gyral substance. Thrombotic softening and abscess formation may be encountered.

The cord is affected in about one-third of the cases. Its meningeal and deeper conditions are similar to those of the cerebrum. The posterior roots seem especially vulnerable, and present marked inflammatory and degenerative changes of their hyaline and axis-cylinder elements. The exudate is usually thicker on the posterior surface of the cord, probably from the usual dorsal position of the patient. This may also account for the preponderance of changes in the posterior part of the cord.

From the gross appearance it is evident in severe cases that meningitis is attended by a certain degree of *encephalitis*. The anatomy of the meninges explains this. The histological changes consist of capillary and vascular dilatation in the pia and an active diapedesis into the perivascular sheaths. These are dilated and crowded with leukocytes and purulent elements. The neuroglial cells and network of the cortex show some proliferation. The *bacteriology* has already been indicated.

The *body signs* in the secondary varieties show the varying lesions of the primary disease, such as tuberculosis, typhoid, pneumonia, infectious endocarditis, or local septic processes. Spinal enlargement may alone mark the infectiousness of the disease.

**Symptoms.**—The *incubation period* of meningitis is an indefinite one. In some epidemics it has appeared to be somewhat less than a week. Usually there is an *incubation period* of several days or weeks, attended by malaise, discomfort, slight feverishness, and headache. More pronounced disturbance then ensues, and we have a *varying period of exuberant*, followed by one of depression, stupor, coma, and death. In the fulminant cases the severest manifestations of the disease are present almost in a moment, and death may occur in twenty-four hours.

The prodromal headache is severe, continuous, and of all the symptoms it is the most constant and significant. It is particularly violent and unmanageable. The patient constantly complains of it, and when stupor or coma has supervened, by holding the head in the hands and by moans and facial expression, he still indicates its often overmastering presence. In children it gives rise to the sharp cephalic cry that punctuates their stuporous state at frequent intervals. Ordinarily it is referred to the occiput or vertex, but is often diffuse.

*Delirium* is common in children and frequent in adults. A mental foggy state is often early noticeable. The patient, roused by the cephalalgia, seems irresponsive, unimpressible, and is hazy in his replies. The delirium is of a low grade generally, but may be wild and frantic or suggestive of the busy delirium of alcoholism and typhoid. Feeding of a projectile character is rarely absent in children, but is less common in adults. The stomach seems simply intolerant and rejects without reason the unchanged ingesta. It may be an early symptom. At the same time the tongue may be quite clean; later it is often thickly furred and suggestive of typhoid. The bowels are usually constipated and the abdomen retracted.

Coxsiveness in the early stages, particularly in children, often occurs. They are general in character and of protracted duration. When the convexity is invaded, they may present a limited distribution, one side of the body, the face, or a single extremity being alone involved.

Almost invariably there is more or less muscular rigidity. In most cases this is marked at the neck by a tendency to retraction that is



Fig. 27.—Albion of patient with meningoencephalitis.

highly significant. At first the patient complains of a feeling of nuchal stiffness and soreness, and finds slight relief in resting the head on a chair-back or over a firm pillow. In the convulsive condition the head is often strongly retracted and the occiput drawn well between the shoulders. When less marked, an attempt to passively bring the head forward will provoke distress and resistance, and at the same time the lower extremities often will draw up sharply (Rodin's sign). A similar rigidity rarely invades the muscles of the lower jaw, producing slight trismus. It may involve the extremities, and when the meningitis has attacked the spine the trunk is often held rigidly in a position of dorsal extension. Kernig and Bull first described a peculiar rigidity in the lower limbs. If the patient is placed in a chair one is unable passively to extend the knees owing to the contracture, which disappears when the thigh is straightened on the trunk.

Kernig's sign can be readily sought with the patient in the dorsal decubitus by raising the lower extremity to a vertical position with fully extended knee. The muscular retraction of the hamstring group, if present, prevents full elevation. The age of the patient must be considered, as in the aged full extension in this posture is not to be expected. Joint disease and deformities must also be excluded. According to Herrick, Osler, and others this sign is practically constant in this disease. It appears early and is certainly valuable. Muscular weakness is usually present and may be more or less localized.

The cranial nerves in most cases sooner or later show invasion and furnish valuable diagnostic symptoms. The olfactory nerves are seldom disturbed, but occasionally the patient complains of olfactory sensitiveness. Photophobia is a common symptom. The optic nerve is irritated by extension of the inflammation down its sheath. After a few days haziness of the disc and enlarged vessels are often seen ophthalmoscopically. The disc may be markedly obscured. Retinal hemorrhages,

<sup>1</sup> "Berlin Klin. Wochensh.," 1894-1895.



papillitis, and subsequent atrophy and blindness are occasionally encountered. Chorioiditis, or panophthalmitis, is sometimes present and may cause great injury to the eye or result in complete blindness. The *third nerve* is almost always affected. When marked strabismus does not demonstrate it at a glance, by having the patient turn the eyes in various directions a lack of conjugate action becomes apparent. Questioning may develop the history of visual uncertainties—haziness or transient diplopia. The *pupils* also furnish important signs. Early in the attack there is a tendency to miosis, which later is replaced by pupillary dilatation. The reflex to light is lost or greatly reduced in amplitude or activity. The pupils may be perfectly immovable. They are often unequal. The *facial nerve* is exceptionally parietic, allowing the face to deviate to the opposite side. At the same time the *auditory nerve* is implicated through its association with the facial nerve within the bony walls of the internal meatus. The loss of hearing that sometimes follows cerebral meningitis is due principally to an extension of the inflammation to the labyrinth, and only exceptionally to destruction of the auditory nerve-trunk. Irritation of the auditory apparatus is apparent in the early stages of the attack. All sudden or loud noises greatly distress the patient. Implication of the *glossopharyngeal* in rare cases causes deviation of the tongue. Probably all other cranial nerves are similarly affected. Doubtless the common respiratory and cardiac disturbances are, at least sometimes, attributable to injury of the pneumogastric.

In addition to the hyperesthesia of the special senses much *tenderness* is customarily found over the spine and limbs. The back of the neck is especially sensitive, and firm pressure of the muscular masses is usually resisted. The entire head is more or less sensitive to deep pressure, and pain is provoked by gentle percussion over the skull. The parietic and sensory disturbances may be unilateral or monoplagic in distribution. The location of the disease causes these variations.

Cerebral meningitis presents no uniform *temperature curve*. It may be ushered in by a chill and an elevation of the body-heat to 104° or higher. It may show a very low, even a subnormal, temperature at first, and terminate with a high range. The evening rise may default. Fickleness is the rule and, in a way, diagnostic.

The *pulse* shows variation in two directions. During the early stage of excitement it is likely to be full, active, and greatly accelerated. Not uncommonly, however, the physician is surprised to find a sluggish pulse of 40 or 60 and the temperature mounting above 100°. This dissociation of *pulse and temperature* is usually manifest sooner or later in meningitis, and furnishes a sign of capital importance. Toward the fatal end the temperature bounds upward, attended by a pulse of great, almost uncountable, rapidity.

*Vasomotor signs* are not lacking. *Herpes labialis* is as common as in pneumonia and as significant. If the finger-nail be drawn across the integument of the abdomen or elsewhere, it is followed closely by a congested red streak that persists for many minutes. Transcendentalists much stress on the phenomenon, which he called the *triple erythema*, and it is of some significance. Taken alone it is of no importance, as it is common to many conditions. The application of mild irritants or gentle heat is



likely to be followed by vesiculation and sloughing. *Urticaria* is often present. A red *arterial* eruption which gives to cerebral meningitis its sometime name of spotted fever is of infrequent occurrence. It appears mainly on the abdomen and trunk, and may be mistaken for the petechial marking of typhoid fever. It is more like roseola.

Early in the attack the respiration may be quickened. Later, in the stupor and coma, it is often slow and irregular. The Cheyne-Stokes variety is often observed, and is of very serious though not absolutely of fatal import. Bot's deep-sighing type of respiration is common in adult cases and undulatory or wave-like respiration is observed. The ordinary relations of respiration to the temperature and pulse may be, and often are, disturbed.

The *urine* shows the general febrile disturbance, and is scanty, high-colored, and heavy. Sometimes albumin and sugar are found in quantities varying from a trace to large amounts. They are significant of irritation of the medullary centers in the floor of the fourth ventricle.

The *feces*—refers more or may not be much modified. Sometimes they are exaggerated, more often decreased. It is not uncommon to find them diminishing as the depressed stage comes on. Their obliteration after the disease has lasted a week or ten days in protracted cases is often noticed. They may be more disturbed on one side than on the other. The toe-sign is frequently encountered.

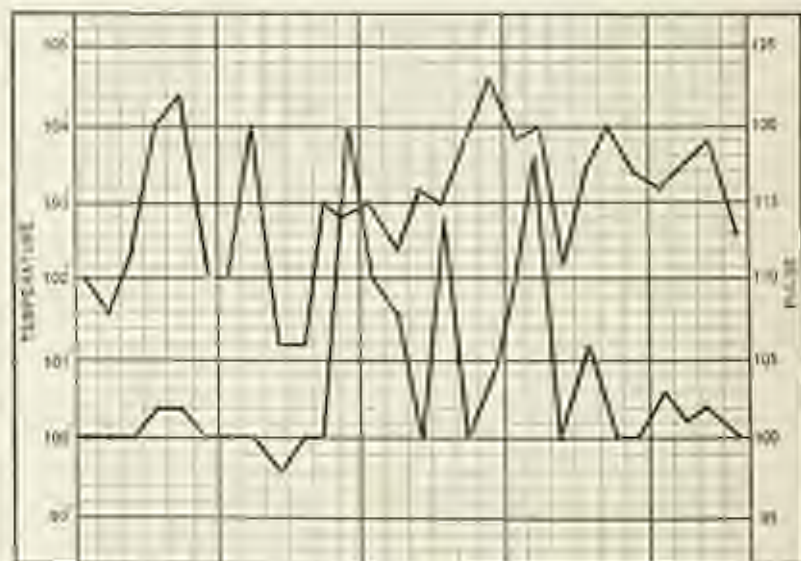


FIG. 28.—Partial respiration and pulse-curve in a case of meningitis of the convexity, showing remission. Temperature, upper line; pulse, lower line. Read twice every four hours, commencing at midnight.

**Course.**—No two cases present parallel conditions as to course, symptoms, or intensity. A fulminant case may end fatally in a day. Protracted cases extend over several weeks and some are clearly sub-acute. An average duration of about fifty days has been noted by Tooth,

Barlow and Lees, and others in the sporadic forms, some extending beyond one hundred days. In the epidemic cases, according to Mallory and Wright, the average duration is about eleven days, perhaps indicating a more virulent variety of the coccus. An ordinary non-epidemic case presents perhaps a week of malaise, a week of excitement, and a week of depression, which usually terminates in deepening coma and death by cardiac failure. At any time, even when the patient seems to be in *artresia*, the symptoms may clear away and convalescence follow. A remission of symptoms may give false hope, to be destroyed by the return of the serious features of the disease in all their intensity. When the basilar region is principally affected, the course of the disease is likely to be cut short by bulbar invasion. The convexity cases are of longer duration and the patient thus affected is sometimes gradually worn out by the pain, delirium, and fever.

During the period of excitement of functions, vomiting, delirium, vertigo, high temperature, muscular rigidity, spasms, convulsions, indeterminate headaches, and hyperesthesia are the prominent features. They are gradually replaced by increasing apathy, stupor, and coma, in which the patient lies inert. He may be nearly relaxed or lie with retracted head, marked spint, and rigid limbs, passing urine and feces under him, and responding to strong stimulation slightly or not at all. The rapid respiration of the first period is replaced by the slow or irregular breathing that indicates increased intracranial pressure or pneumogastric irritation. Finally, deglutition is impaired, respiration becomes stertorous, and the patient slowly sinks or may expire in a convulsion.

Various epidemics have shown numerous features of similarity among the cases that have marked their progress. At first the fulminant cases have been common. At the end these of less and less severity are met with.

Diagnosis in the very early stage is difficult. When the disease is fully developed, and in epidemic conditions, a mistake would be almost impossible. No one symptom is constant, and it is a disease of protean aspects. The symptoms vary as the base or convexity is involved. Meningitis of the convexity is marked by excessive delirium, high temperature, convulsions, localized spasm, dyesthesia, and monoplegia. The cranial nerves are not involved, and ocular symptoms are slight or entirely wanting.

When the base is affected, the temperature often has a low range or is subnormal. Cranial palsies are common, retraction of the head is nearly always present, and the dissociation of pulse, temperature, and respiration is marked. Optic neuritis declares the basilar location or extension of the disease. As a rule, infection by the blood-channels results primarily in meningitis of the convexity. In rare instances the meningitis is confined to the ventricular area and the choroid appendages, but no distinctive symptoms indicate this limitation. The diagnostic value of the headache depends upon its duration, intensity, and particularly upon its persistence after delirium has appeared or stupor has ensued. The tenderness of the head and the pain on pressure are of some value. Vomiting is only significant when of the projectile character, unattended by gastric disturbance and fermentative changes. The temperature lends aid when out of keeping with the pulse-rate and



respiration rhythm. The tenderness in the limbs, the rigidity of the neck, the contractures at the knee shown in Kernig's sign, are highly suggestive. Apathy, drowsiness, and mental obscurity in themselves should suggest the disease. The grouping of several of these symptoms would justify a tentative diagnosis, which the appearance of squint, convulsions, delirium, optic neuritis, Kernig's sign, or the vasomotor symptoms would confirm. Gowers states that in suspected cases the too free vesiculation of the skin under heat or irritation would lend support to the diagnosis of meningitis. *Lumbar puncture* will also assist. In practice it should always be done. It furnishes, through bacterial tests, knowledge of the exact infection, often establishes the diagnosis, and sometimes contributes to beneficial treatment. In the diplococcus variety it gives us exact indications and the opportunity for the use of Flexner's serum. In meningitis the spinal fluid is usually turbid and may even be puriform, though it may be quite clear even when the cerebral membranes are covered with a purulent exudate. In tubercular meningitis it is generally clear and inoculation experiments are often required to demonstrate the tubercle bacilli. The cellular content is always increased.

Differentially, the diagnosis is often much complicated by the association of meningitis with other affections which may overshadow it. As it occurs in the course of pneumonia, typhoid fever, cranial injuries, septic invasion, and pyemias, the original disease process may entirely absorb the practitioner's attention. From typhoid, Hirt<sup>1</sup> says, it is sometimes indistinguishable, presenting a similar temperature curve, splenic enlargement, rose spots, and the typhoid stool. Widal's reaction is a helpful but not absolute sign of typhoid. The presence of postmeningeal symptoms in pneumonia of the apices has been mentioned, and in pneumonia generally meningitis can only be determined by lesser symptoms. Uremia should be excluded by a thorough urinalysis. An attack of delirium tremens may be easily mistaken for meningitis, with which it is not rarely complicated. Single or double acute arthritis in children, marked by constant pain and vomiting, delirium, and deafness, is usually confounded with meningitis. If the facial nerve escapes, the deafness would point to labyrinthine disease and serve to exclude meningitis. Hysteria is sometimes mistaken for meningitis. The emotional and mental features, the normal temperature, bounding, and pulse, and certainly the stigmata of hysteria should differentiate it. In children the onset of nearly every febrile disturbance has been confounded with meningitis. Here reliance must be placed on the characteristic temperature curves, eruptions, and clinical antecedents. The character of the infection can usually be determined by spinal puncture and appropriate bacteriological tests.

**Prognosis.**—In expressing an opinion as to the outcome of a given case, the physician has three positive statements to make. First, the result is absolutely uncertain until death occurs or convalescence is firmly established; second, the probability of a fatal termination is always pronounced; third, some trace of the disease is likely to remain permanently. The disease is full of surprises. The writer has seen a patient in deepest coma for hours, with convergent squint of the

<sup>1</sup> Hirt, "Diseases of the Nervous System," New York, 1893.



most marked variety, retracted head and abdomen, convulsed limbs, Cheyne-Stokes respiration, large quantities of sugar in the urine, and a pulse of 32, entirely recover. Again, cases that seem trifling or on the high road to recovery suddenly become worse and die. Purulent cases are practically always fatal. If a purulent focus within the skull, nose, ear, or throat is found, it renders the outlook extremely grave. A meningitis following pneumonia is practically fatal, but Stoelzner<sup>1</sup> reports a recovery where spinal puncture showed both pus and the pneumococcus. The cerebrospinal form runs a more favorable course, the mortality varying from about thirty to sixty per cent. in various epidemics. Partial recoveries, however, greatly outnumber cures. The extension of inflammation to the labyrinth produces deafness, which in young children may lead to deaf-mutism. Optic neuritis is followed by dimness of sight or complete blindness. Local meningeal thickenings and cerebritis portend monoplegia, epileptoid convulsions, and mental defect. Though the disease is not one of proved contagiousness, separation of the patient from the other members of the family should be insisted upon. The tubercular variety is almost invariably fatal.

**Treatment.**—The first consideration in the management of a case is the removal, if possible, of the infection stratum. Cranial suppuration and injuries demand immediate surgical attention. Optic, nasal, pharyngeal, intestinal, and pelvic cavities should be rendered aseptic if their organs or contents fall under suspicion. Intestinal antisepsis is always in order. To this end free catharsis and the administration of antifermentatives are indicated as well as by the usual constipation. Minute doses of calomel,  $\frac{1}{8}$  of a grain, repeated every half hour or hour until active results are obtained, can be highly recommended for this purpose. In addition, the mercurial may have a widely diffused microbicidal value.

From the first the patients should be kept in a cool, quiet, moderately darkened room, and all annoyances to which their hyperæsthetic senses make them especially responsive should be prevented. Extreme delirium will often require a sedative. In the presence of the cephalalgia bromide, chloral, Indian hemp, and other ordinary means will practically always fail. The coal-tar derivatives are equally valueless, and nothing but morphia will give relief. Its hypodermatic administration is by all odds to be preferred except in infants. If high temperature be present, antipyretic drugs must be employed with great circumspection. They often have no effect, and their depressing action sometimes constitutes a real danger in a disease marked by symptoms of disturbed cardiac and respiratory innervation. Tepid to cool baths with cold affusions to the head, sponging, and the pack may be used to better advantage. Hot baths are decidedly valuable. They often allay the delirium, reduce the temperature, and clear up the clouded mind. The use of an ice-bag or ice-coil to the head frequently gives comfort to the patient and renders the head-pain bearable. Counterirritation along the spine is a measure of doubtful utility, and always attended by the danger of setting up serious ulceration or actual sloughing.

In the depressed period sedatives are no longer required, and stimulants, like strychnin and whisky, will often need to be sharply pushed.

<sup>1</sup> *Berlin Klin. Wochens.*, April 19, 1897.

to meet the failing action of the heart or lungs. When coma has appeared, it may often be broken and sometimes happily ended by the application of a fly-bliste to the nape of the neck. It should be sufficiently large,—two by six inches for an adult,—and extend from one mastoid to the other. If this is not efficacious or the coma again develops, a drastic cathartic—one to five drops of croton oil in emulsion—may render similar service. In the same way repeated spinal puncture with full drainage of the fluid sometimes produces immediate benefit. By recurring in turn to the blister, cathartic, puncture, and baths, with cool affusions to the head, no doubt the progress of the disease may be often hindered. Unfortunately, it usually again takes up its course, but occasionally such measures seem to definitely check the malady and recovery slowly follows. Aufrecht<sup>1</sup> recommends full hot baths of about 100° F. for ten minutes. Their apparent effect sometimes is to reduce temperature, diminish the headache, accelerate the pulse, and induce sleep. As many as eight a day have been given with excellent results by Worochilsky.<sup>2</sup>

The use of large doses of iodids with the expectation of causing resorption of the effusion usually only results in distressing the patient's stomach, reducing his strength, and increasing the vomiting. Shaving the head is rarely called for, but a heavy mass of hair may usually be removed with advantage. Mercurial inunctions seem at times to lead help even in the absence of syphilitic taint. It is immaterial whether they be applied to the scalp or to the limbs, so far as the effect is concerned, but the non-hairy parts are more convenient and less irritable. Credé's collod of silver ointment and iodoform ointment in large doses applied to the shaven head have advocates, and may be properly employed. Good results have been claimed for subcutaneous injection of the bichlorid of mercury in doses of  $\frac{1}{16}$  of a grain several times a day in children. In cases showing the diplococcus of Weichselbaum, Flexner's serum should be employed by spinal puncture. Favorable results from its use seem to be directly proportional to its early administration. Every suspected case must, therefore, be subjected to spinal puncture and bacteriological examination as an initial proceeding.

Important items in the treatment of meningitis are the nursing and nutrition of the patient. A careful, intelligent, preferably trained, nurse who will exclude visitors and members of the family and prevent disturbing sounds, bright lights, and all annoyances, is the salvation of some cases. Thus only can constant watchfulness of pulse, respiration, and temperature, which may show extreme and critical variations in an hour, be provided and emergencies met as they arise. Nourishment in small quantities can usually be administered frequently. If the cerebral vomiting persist, nuchal is practically the only measure we can rely upon to check it. In the stuporous states rectal alimentation will be serviceable, or the nasal stomach-tube in skilled hands may be employed. If deglutition is difficult, one of these is imperatively demanded. Large quantities of soups, custards, whey, junket, beef-juce, and eggs may be given with advantage from first to last. The bowels must be active.

The question of trephining the cranium and draining the meninges and ventricles has received favorable consideration in those cases where

<sup>1</sup> "Therap. Monatshefte," Aug., 1894.

<sup>2</sup> *Ibid.*, Feb., 1895.



deepening coma and failing circulation point to increasing intracranial pressure. The results thus obtained, however, are not encouraging.

In recovered cases treatment is directed toward building up the general health. Inflammatory thickening and remaining effusion may perhaps be benefited by iodid of potassium and mild mercurial courses. Weakened or paralyzed extremities should receive careful electrical and massage treatment as soon as the active febrile stage subsides. All severe physical and mental fatigue should be avoided for a long period. Exposure to the sun and the use of stimulants will be found especially detrimental.

#### VARIETIES OF LEPTOMENINGITIS.

**The Diplococcus Variety of Meningitis.**—In the description of leptomeningitis which has preceded, the *epidemic* and *sporadic* features have been made prominent, and cases due to the diplococcus infection have been the basis of the descriptions.

**Etiology.**—In this variety the etiology of the disease is clear and definite; it invariably is due to the invasion of the diplococcus of Weichsellmann, which is very commonly found in pure culture in the membranes and ventricles of the brain, or in the fluid obtained by spinal puncture. It is probable that the germ finds entry through the respiratory spaces, either by way of the nose or the lungs. Its well-known endemic and epidemic character furnishes one of the most serious diseases under circumstances where many people are compelled to live in narrow quarters—as in barracks, on shipboard, in schools, etc., but where sanitary precautions can be maintained and disinfection practised it is apparently readily controllable. The detection and control of carriers is highly important, but, as yet, the complete eradication of infection in such cases presents very serious difficulty.

The **symptomatology** embraces features which have been dwelt upon in previous pages. The onset, usually insidious, with headache and malaise, sometimes is abrupt.

The **course** of the disease varies from a few days or weeks to even a few months, and in various epidemics shows a similarity of features, the early cases being more severe than those that develop later, so that toward the end of the outbreak the severity of the disease seems to be attenuated and the prognosis improved.

**Diagnosis** of this variety rests upon the recognition of the bacterial infection, and this is obtained usually by spinal puncture. While one may always suspect the diplococcus in epidemic conditions, the proof is easily obtained.

The **prognosis** varies under different conditions of a given epidemic, and in different epidemics it also shows a considerable range of mortality percentage. On the whole, the prospects are better than in any other variety of meningeal infection, from 30 to 50 per cent. of cases recovering.

The **treatment**, while embracing the general features of nursing and care which have been already outlined, must be said to turn specifically upon the evacuation of a certain amount of spinal fluid and the injection of a certain amount of Flexner's serum. The usual dose for an adult is 40 c.c. By spinal puncture a similar amount of spinal fluid is



evacuated, and immediately the serum is introduced into the spinal canal. The same or a somewhat smaller dose is repeated in twenty-four or forty-eight hours, depending upon the progress of the case; and again later on one or more doses may be used if required. The percentage of recoveries under this administration, when early employed, is decidedly greater than under any other method of management. The patient should be treated as an infectious case, measures of isolation maintained to guard those who might otherwise come in contact, and in most large cities it is required to report cases of this sort to the health officers.

The tubercular variety of meningitis, *basilar meningitis*, acute *hydrocephalus*, is a local manifestation of the action of Koch's bacillus, usually most intense at the base of the brain, commonly attended by distention of the ventricles, almost invariably if not always secondary, and practically always fatal.

**Etiology.**—The pathological process in this form of meningitis is clear and definite. The bacillus of tuberculosis carried to the meninges of the brain by the vascular system finds in the pial fluid and the perivascular spaces a suitable nidulum and an excellent breeding ground. In the great majority of cases a primary focus of tubercular infection is found in other organs. The absolute exclusion of such original disease is practically impossible in the few remaining cases where it is not readily discovered. It has been suggested that infection might reach the cranial contents by way of the nasal vault and cribriform openings. This can not be denied, but seems improbable and lacks confirmation. Though tubercular infection of the soft brain-coverings may occur at any age, the years of life between two and ten show an overwhelming proportion thus affected. Before the age of six months and in advanced years it is practically unknown. It gradually diminishes after the age of ten and is infrequent after thirty-five. The other *etiological factors* are those that are common to all tubercular processes. Heredity, both tubercular and septic, has a significance. Urban populations, conditions of crowding, winter and spring (the seasons of exposure and poor ventilation), depressing influences, deprivation, and tubercular contamination are included in the list of predisposing and exciting causes. Traumatism may induce a tubercular meningitis perhaps by reducing the resistive factor in the tissues that stays the bacillary invasion. At any rate, it is a common observation that spinal and head injuries in themselves rather trifling, are followed by the disease in numerous instances.

**Pathological Anatomy.**—Upon opening the skull and reflecting the dura, hardly any inflammatory action is, as a rule, to be seen. The arachnoid is sometimes a little lacking in luster along the course of the main cerebral arteries. At the base, however, the mottled picture is comparatively uniform and striking. At the arterial circle of Willis, extending along the basilar furrows, between the peduncles and the pons, covering the interpeduncular space, and especially marked in the Sylvian fissures, is a thick, almost gummy exudate, dotted with small masses of a dirty whitish color. These are tubercles in various stages of development or degeneration. They frequently dot the pia on the lateral aspects of the brain and sometimes reach to the vertex. They are always grouped near the blood-channels and vascular spaces, through which evidently the infecting organism reaches the meninges.

The exudate, often of a clear, jelly-like consistence, is frequently turbid, grayish-yellow, and not rarely purulent or even greenish. It is found in greatest quantities at the base, but ascends with the cerebral vessels along the pial spaces, filling the sulci and interlobular grooves. It travels down the perivascular sheaths into the cortical substance, and invades the ventricles through the transverse fissure. The inflammation is here propagated to the ventricular ependymal lining, resulting in a great increase of fluid, with dilatation of the ventricles and a condition which early gave to this disease the distinctive name of *acute hydrocephalus*. This feature is seldom wanting and may, by pressure, cause marked flattening of the convolutions.

The granular tubercles, usually visible at a glance, sometimes require a little search. If the pia be stripped off and floated in a little clear water, the tubercles can be much more readily distinguished. They are grouped about the arterioles or disseminated along the larger vessels in patches and small masses, which may rarely unite to form a continuous covering for wide areas of the brain-surface. They present the variations which mark tubercles elsewhere, depending upon their age and development or disintegration. Microscopically, they are often found to occlude the perivascular spaces, giving rise at times to small softenings and hemorrhages, which occasionally attain sufficient proportions to explain the focal symptoms that may have been clinically manifest. These infarcts are usually found in the basal ganglia and the cerebral peduncles. The perivascular extension of the inflammation determines more or less *cerebritis*, and when the pia is removed it often decorates the brain in consequence of the soft adhesions that have formed. Infrequently there are slight adhesions between the pia and dura.

In a large proportion of cases of tubercular meningitis the spinal cord is also invaded. The meninges, meningeal vessels, and the cord itself present features analogous to those found within the cranium. The exudate is most marked in the anterior and posterior grooves of the cord, and the meningeal changes are always greater on the dorsal surface.

Tubercular meningitis presents varieties in which the pathological findings are most marked at the vertex. This is so rare that the disease was formerly known and described simply as basilar meningitis. Occasionally a circumscribed tubercle or a tubercular mass gives rise to symptoms of, and constitutes, an intracranial tumor. Again, the meningeal involvement is only a part of disseminated miliary tuberculosis, appearing at once in the head, lungs, intestines, peritoneum, and abdominal parenchymatous organs. In these cases, while all the pial structures are the seat of granular tubercles, they are most profuse along the vascular routes. Ordinarily, they are not attended with much exudation and brain symptoms may be quite lacking during life.

Bacteriologically the tubercle bacillus of Koch is always found, and usually is present alone. Secondary mixed infections are extremely rare and purely adventitious. A case has been met in which the pneumococcus was also present. Primary tubercular processes in other organs, as in the serous sacs, the lungs, the intestines, the mesentery, the mediastinum, or the genito-urinary tract, are commonly found. A single caseous mediastinal gland has served as the starting-point for the meningeal infection.



**Symptoms.**—The *onset* of tubercular meningitis is insidious and its course protracted. Cases, however, manifest wide variations. The typical cases occur in childhood. Those occurring later in life are usually preceded by well-marked tubercular disease to which the meningeal complication is added in a natural order. In adults transient peculiarities of a mental character, such as lethargy, anorexia, childishness, and irritability, may be prominent for weeks before distinct meningeal symptoms appear. Children, on the other hand, frequently present an antecedent appearance of good health, the primary focus of disease having been so insignificant as to produce no notable symptoms. They become peevish, fretful, and out of sorts. Appetite and sleep are disturbed. After a week or two of protracted malaise with perhaps a little headache, an occasional vomiting spell, and slight febrile disturbance, they are noticed to be apathetic, then distinctly drowsy, and later stuporous. If disturbed, they complain of headache or manifest discomfort and may vomit. The triad of symptoms—*headache, vomiting, and constipation*—following a more or less protracted period of malaise, which may rarely extend over months, marked by general physical deterioration and often by great loss of flesh, has serious significance. The stuporous repose is often pierced, but not broken, by a sharp cry of pain, the *hydrocephalic cry*, which in some cases is frequent and distressing. It is apparently due to the head-pain. There is retraction of the head with more or less *rigidity* of the neck, and the entire spine may be fixed. Slight opisthotonos is common. The thighs are flexed on the trunk, the legs on the thighs, the abdomen becomes more and more retracted, and finally presents the scaphoid or boat-shaped hollowing that is classical. The masticatory muscles sometimes are similarly stiffened, and the Kernig symptom of rigidity of the knee when the hip-joints are flexed is usually present.

The *head-temperature*, sometimes indistinct, are often increased at first and gradually diminish as the depression and stupor develop. The *respiration* continues at intervals and is of the cerebral type, unattended by evidence of gastric disturbance. The *temperature*, though fickle, usually shows an evening elevation. It is rarely extremely high until the final termination of the disease, when it attains  $103^{\circ}$  to  $105^{\circ}$  F., or even more, but from  $101^{\circ}$  to  $102.5^{\circ}$  is frequently seen. Occasionally it drops below the normal and may show considerable variation in the course of an hour.

The *respiration* is not notably disturbed until stuporous or comatose conditions obtain, when it is irregular, sighing, slowed, and of the Cheyne-Stokes variety; but Simon<sup>1</sup> asserts that from the first there is a lack of harmony in the respiratory movements of chest and diaphragm. The *pulse* then becomes much altered. It is slowed, often irregular, and just before death becomes uncountably frequent. It is in tubercular meningitis, especially that the *dissociation* of pulse, temperature, and respiration is found. Its diagnostic value is great. Thus, when the temperature exceeds  $100^{\circ}$ , the pulse may show a subnormal rate, and the respiration be slow or rapid.

As the lesion is usually basilar, involvement of cranial nerves is the rule. Sight is often dimmed, and the ophthalmoscope demonstrates

<sup>1</sup> "La France Méd." March 20, 1895.



changes in the fundus-picture in a majority of cases. Simple hyperemia, marked congestion, and papillitis may be expected. Of pathognomonic importance is the not infrequent presence of tubercles in the retinal or choroid tunics. Very early there is disturbance of the *third nerve* in the form of papillary inactivity and a tendency to miosis, which later gives way to wide dilatation. Squires<sup>1</sup> describes a rhythmical dilatation and contraction of the pupils caused by extending and flexing the head. As the head is bent backward the pupils slowly dilate and again contract when the head is brought forward, the pupillary variation being proportionate to the amount of flexion and extension. Strabismic deviations of the eyes, or fixity of the globes, should be carefully sought for. In older patients diplopia is frequently noted. A strong convergent upward squint, drawing the pupils almost to the inner canthi and turning the globes well up is usual in deep coma. The *facial nerve* is rather frequently affected with a corresponding parietic condition of the face. Deviation of the tongue, pharyngeal and laryngeal distress, mark the implication of the posterior members of the cranial group. The *vestibulocochlear*, aside from the stupor, is one of confusion, often marked by moderate delirium. When the patient is roused or is able to respond he presents all interference. Rapid *excretion* attends the progress of the disease. *Sphincter control* is not often disturbed, except that in the comatose state the bladder and bowels act automatically, and their contents are unconsciously voided under the patient. Retention of urine, however, is sometimes noted and persistent; *obstinate constipation* is the rule unless intestinal tuberculosis causes diarrheal discharges.

Vasomotor disturbances are manifest in flushes and pallor, which frequently succeed each other rapidly or appear side by side on face and trunk in peculiar distribution. The *tortic colliculus* is present. The persistence and intensity of the streaks on the skin and the ease with which they are produced give some significance to a phenomenon observed in many smallpox eruptions.

Convulsions sometimes open the sequence of acute manifestations, but more often appear later; very rarely are they entirely absent. Due in large part to the irritation of the bulbar region, they are usually generalized and protracted. Convulsive *twitchings*, especially in the face and hands, indicate the extension of the irritation onto the lateral aspects of the brain. By localized disturbances in the motor cortex, *epilepsy* of a Jacksonian variety may be induced. Similarly paresis is often found, which may be localized. In cases of protracted onset, some wavering in the gait, or even marked staggering and clumsiness, are of the same significance.

After a variable, active period of a few days, a week, or even longer, a marked *remission* of the stupor, convulsions, vomiting, and other signs of active disease is usually noted. It is frequently followed by a period of vacillating improvement that only too often gives false hope and ill-founded security. After a number of oscillations, in some of which the patient may seem to touch the border-land of complete relief, the disease again takes up its course to a fatal termination. Such remissions may last from one month to a year,<sup>2</sup> and terminate without cause,

<sup>1</sup> N. Y. Med. Rec., March 26, 1901.

<sup>2</sup> E. H. Lee, "Thèse de Lille," 1904.

or apparently as the result of a fall, shock, or intercurrent slight illness. All the symptoms of depression reappear, the coma intensifies, the pressure indications increase, the temperature runs up, stertor comes on, the pulse is inordinately accelerated, and death from respiratory or cardiac failure closes the scene, often attended by convulsions, due, perhaps, to the asphyxiated blood state. Occasionally a stuporous or even comatose condition, lasting days and weeks, may precede the fatal termination. *Spinal symptoms* are common. The rigidity of the back and lower extremities, and great tenderness along the spine and over the skin, indicate spinal involvement; but the overwhelming cerebral side of the disease usually obscures the less strongly marked features of spinal extension.

**Course.**—A disease marked by such pronounced variations of intensity, and even of localization, necessarily presents a lack of uniformity in its course and duration. Most frequently it is subacute. Rare cases reach a fatal termination in a week or ten days after the onset of marked symptoms. On the other hand, the prodromal stage may extend over weeks and months, with remissions of all symptoms at intervals. Again, the mid-period, marked by fluctuation, may protract the disease for days and weeks. Ordinarily, the malady presents four fairly marked clinical stages: (1) The prodromal period, of indefinite length; (2) the period of irritation and excitement, varying from a few days to a week; (3) the period of oscillation, lasting about as long, and, finally, (4) the period of marked depression, attended by paralytic features, deepening coma, and death. This refers principally to the disease as it occurs in childhood. Tooth noted an average duration of fourteen days in 29 cases, of which the shortest was of five and the longest thirty-three days; yet exceptional cases run through many months. In adults it is likely to run a more varied and protracted course, and in the rare senile cases it often provokes but insignificant disturbances.

The diagnosis is avowedly difficult. In the prodromal period it is never positive. When drowsiness, headache, vomiting, and constipation are found following a week or more of malaise and petechiae, the meningeal character of the trouble would be pertinently suggested. The detection of a primary tubercular process, or of tubercles in the retina, is a practical confirmation of the diagnosis. Strong hereditary tendency to tuberculosis, and more emphatically a family history of several cases of tubercular meningitis, would raise a strong presumption, when attended by the cerebral triad, headache, vomiting, and constipation, that tubercular meningeal infection had taken place. Continued exposure to tubercular infection has some significance. The differential diagnosis from acute meningitis depends principally on the subacute onset and lower grade of intensity of all the symptoms in the tubercular variety. This is manifestly indefinite, and clinically the two forms of meningitis can not always be distinguished. Quincke's puncture furnishes a practical test. The bacillus tuberculosis is found in the spinal fluid in about four-fifths of the tubercular cases. Positive inoculation tests in guinea-pigs are equally valuable as proving the tubercular character of the meningitis, even when no bacilli can be found in the fluid by immediate microscopical search. In other cases the presence of the pneumococcus or of pus has made a differential diag-



rosis possible. It is probable that observations of the opsonic index may be of some diagnostic importance here, as in pulmonary and other widespread tubercular processes. The positive tuberculin (von Pirquet) and ophthalmic tests have a value only as showing the presence of a tubercular process somewhere in the body.

**Prognosis.**—Practically the only hopeful point in prognosis is the possibility of a mistaken diagnosis. A few undoubted cases of tubercular meningitis have recovered, and the post-tubercular lesion has been found after death from subsequent and unrelated causes. Dr. G. Fatterer,<sup>1</sup> formerly assistant to Rudolfeisch, relates a case in which tubercular meningitis had been diagnosed by Prof. Leube. Five years later calcareous tubercles were found in the spinal meninges. Hensch, Pollitzer, Freyhan, and Aranzino<sup>2</sup> also report cases that have a similar bearing, and A. E. Martin<sup>3</sup> has been able to tabulate 20 undoubted cases recorded since 1894. Their extreme rarity hardly invalidates the rule of fatality. Owing, in some cases, to the difficulty of a differential diagnosis, the physician should always maintain a margin of reserve. This is particularly important in the oscillatory period of the disease, when apparent recovery may be most deceptive.

**Treatment.**—The same general management and care should be employed as in acute meningitis. Actuated by the beneficial results of iodoforn injections in tubercular joint-disease, in Germany injections of the shaven scalp with the same medicament have been warmly advocated. They may be tried. All are united on the use of calomel in small repeated doses. The mercurial not only is the best agent against the constipation, but may have some effect upon the inflammatory process within the skull. The extreme tendency to rapid emaciation suggests the most careful and persistent efforts to maintain the nutrition by every possible means. When the stomach is intolerant, rectal alimentation may be employed and cod-liver oil injections are useful. Digestion can sometimes be assisted by peptonizing and predigesting the food. Stimulation by whisky or a good wine is important in the later stages. During the period of excitement sedatives are required. In children the bromid of potassium is a useful remedy. It frequently controls the headache and reduces the convulsive tendency. The action of the potassium salt in producing arterial contraction is increased by the addition of chloral, which may be profitably combined with it. Applications of ice or the cold coil to the head seem at times to give some comfort and relief from pain. Antipyretics, aside from frequent gentle sponging, are rarely indicated and usually do no good. The hot bath often effectually controls the convulsions. Of late the value of drainage or more exploration of the peritoneum in tubercular infection of that cavity has turned attention to the possible value of a similar procedure in tubercular meningitis. Some of these cases have been drained and the ventricles tapped with alleged temporary improvement. Quincke's lumbar puncture, which certainly reduces intracranial pressure and removes the fluid, has, in the cases reported by Strahan, been

<sup>1</sup> "Chicago Medical Recorder," June, 1905.

<sup>2</sup> "Rif. Med.," Aug. 20, 1905.

<sup>3</sup> "Brain," 1909, p. 209.



followed by recovery once. As a fatal ending is the only reasonable expectation when the diagnosis has been confirmed by the lumbar puncture, the injection of iodiform emulsions or other solutions within the spinal and cranial dura may be attempted with propriety. Serum treatment to improve the opsonic condition may be properly employed. In protracted cases tuberculin may be used in very small amounts, as there is supposed to be danger of producing a severe meningeal reaction, but such has not been my observation.

**Serous Meningitis.**—A number of cases were first reported by Hugenin, Oppenheim, Eichhorn, and others presenting merely a very much increased cerebrospinal fluid, with a clinical history of a low grade of meningitis. Later, Quincke, by his spinal puncture, was able to make the diagnosis of this variety of meningitis, which he believed to be analogous to a pleurisy, and denied that it was of bacterial origin. In some cases the fluid distends the ventricles especially, and such have been called *meningitis interna serosa*. In other cases the external meningeal spaces were more affected, and the cortical pia, giving rise to the term *meningitis externa serosa*. British authors have apparently described the same condition under the term *chronic infantile meningitis*.

In the etiology of the condition, though Quincke saw only vaso-motor or angioneurotic activity, involving mainly the choroid plexus or the cortical pia, many cases have been recorded in which the serum accumulation was undoubtedly secondary to infections. Finkelstein and Pfundler have reported the presence of bacteria in the cerebrospinal canal. A preceding pneumonia or typhoid is a common clinical observation, and even tuberculosis. A serous meningitis may arise from an otitis media, and in nursing children follows in some instances a gastro-enteritis. In older children whooping-cough and measles, in adults trauma and alcoholism, have seemed to act as causative factors. The infective bacteria presented by such cases embrace pneumococcus, staphylococcus, streptococcus, bacterium coli, tubercle bacilli,<sup>1</sup> and typhoid bacillus. They seem to present but slight virulence, and are found in very small numbers in the exudate of the meninges or of the spinal fluid.

The pathological anatomy of serous meningitis is marked by a flattening of the convolutions and great widening of the ventricles when the fluid is mainly accumulated in these cavities. The fluid is greatly increased in quantity and perfectly clear in appearance. The brain is edematous. The cortical pia generally presents evidence of slight inflammation and a swollen edematous condition. This is particularly marked in those cases where the accumulation of the fluid is mainly exterior to the ventricles. The lymphocytes are relatively few in the cerebrospinal fluid, the albumin increased in quantity.

The symptomatology is neither so severe nor so typical as in ordinary acute infective meningitis. In nursing children, where the disease generally is secondary to intestinal disorders, it frequently is overlooked, but it may present very severe and rapidly developing symptoms, with elevated temperature and convulsions, leading to coma, rigidity of the neck, Kernig's sign, changes of the pupil, and death in a few days. In

<sup>1</sup> Houser, *Bull.*, "Neurology, Constantin," No. 21, 1909, p. 1184.

older children symptoms vaguely referable to the meninges are commonly presented for a long period, with suggestions of brain pressure. Fever, disturbances of vision, modifications of consciousness, slow and irregular pulse, Cheyne-Stokes breathing, generalized and localized convulsions are encountered. In many cases the condition has been mistaken for brain tumor. Choked disc, headache, vertigo, vomiting, convulsions, and palsy of cranial nerves may all be present and make the differential diagnosis very difficult. In some cases serous meningitis follows continuously upon congenital hydrocephalus. In others, through remissions and intermissions, the clinical history extends over years.

The course of the disease is various. Serous meningitis occurring in nurslings is usually of fatal termination, but recovery is not to be entirely excluded. The disease may run weeks and months and eventually in a chronic hydrocephalus, or this may develop later on in life, and some cases show a predisposition to subsequent acute attacks at long intervals during life in consequence of excesses, trauma, infectious diseases, and similar disturbing elements. Some cases of chronic hydrocephalus developing in adults are due to this condition.

The diagnosis is principally to be made by lumbar puncture. A large quantity of fluid is easily obtained, making its exit under high pressure. It is perfectly clear, but rich in albumin. A centrifugal specimen shows but few lymphocytes, and few or no organisms are to be found. Sometimes only by animal inoculation can the bacteriology of the fluid be determined. Chlorosis, sinus thrombosis, and uremia, which all cause increase of fluid and increase in the pressure under which the fluid is to be obtained, may offer complications in the diagnosis, so that after all, the clinical history must be depended upon very largely in making the diagnosis.

Prognosis includes the probability of spontaneous cure, favored by repeated lumbar puncture. This takes place in a manner quite similar to that presented by a pleurisy with effusion, or a hydrocele subjected to repeated puncture. Quincke insisted that cases presenting one-half of one per cent. of albumin or less furnish a good prognosis.

The treatment is already indicated. In making the spinal punctures it is important to withdraw the fluid very slowly, in order that the intracranial pressure be not too rapidly reduced. As much as 100 c.c. is sometimes withdrawn with benefit, and a repetition of the spinal puncture is indicated by a recurrence of the pressure symptoms. Even puncture of the ventricle has been used with advantage and success. In the more acute cases the ordinary treatment of meningitis is indicated.

**Chronic Leptomeningitis.**—Leptomeningitis, as already mentioned, is occasionally subacute. This form may become chronic. As the result of alcoholic excesses, syphilis, and involution, sometimes a low grade of chronic leptomeningitis is developed. Post-mortem examination frequently reveals such a condition which may have been unsuspected during life. Among the chronic insensit it is a very common finding after death. Clinically, except in the syphilitic form, its manifestations are very uncertain and obscure. Stiffness of the neck and persistent slight headache, both marked by exacerbations, with some



tenderness over the skull, may be complained of. Undue optic and auditory sensitiveness usually accompany the more severe periods of headache. All causes of cerebral congestion, such as stooping and muscular or mental exertion, cause distress and intensify the headache.

The alcoholic cases are the least well marked. A low grade of optic neuritis may be present, and usually disappears if the alcohol is withdrawn. Slight mental cloudiness or delirium, due to the specific action of the poison on the brain-tissue, is frequently observed. Multiple neuritis and other indications of alcoholism are usually present, and often quite overshadow the meningeal symptoms.

The syphilitic inflammation is usually circumscribed, and gives rise to local symptoms. It will be fully considered under the head of Syphilis of the Nervous System, Part VI.

### CHAPTER III

#### DISEASES OF THE FIRST AND SECOND CRANIAL NERVES.

DISEASES of cranial nerves are broadly divided into those which affect the *cortical centers*, those which involve the *nuclei*, and those which involve *trunks and peripheral portions*. The peripheral nerves extend from the nuclei to the ultimate distribution of their filers. A part of their course lies within the mass of the brain itself. While the peripheral portion of a cranial nerve may suffer independently of its nucleus, injury to the nuclear center is always followed by degeneration in the peripheral part. It is not unusual for a peripheral nerve to be singly injured by traumatism, or even by disease, but, owing to the close anatomical and physiological relations of the cranial nuclei, the medullary centers are rarely individually diseased. Very often nuclear disease of the cranial nerves is but a portion of a more general nervous malady. The successive nuclear centers in the medulla, along the floor of the fourth ventricle, under the aqueduct of Sylvius, and on the posterior wall of the third ventricle, constitute the upward prolongation of the spinal gray matter. They may be involved with the spinal centers at the same period or at varying stages of a given general malady. The peripheral parts of the cranial nerves also participate in the general diseases which affect the spinal nerves and react similarly to infections and poisons. The analogy of the last ten cranial pairs to the spinal nerves should be clearly apprehended. The olfactory and optic nerves react more, as does the brain proper. They are, in fact, portions or lobes of the brain. To a less degree the same is true of the auditory nerve.



**Diseases of the Olfactory Nerve.**—The exact cortical origin of the olfactory nerve is not known. Its pathway in the brain is not clearly traced. Its disturbance, usually considered of slight importance, is often overlooked. Ordinarily, the olfactory cortical center is assigned to the uncinate gyrus, or near by, in the cornu ammonis in the floor of the lateral ventricle.<sup>1</sup> Certain cases of epilepsy in which an aura referable to the sense of smell was noted have presented post-mortem evidences of disease of the temporo-sphenoidal lobe near to or involving the uncinate convolution. The same is true of some cases of mental disease marked by hallucination of smell. It seems probable that the olfactory nerve is represented on both sides of the brain.

A loss of smell on the side opposite to a lesion in the posterior portion of the internal capsule has been noted by Férri<sup>2</sup> and confirmed by others. Usually, however, the lesion is on the same side of the brain, and involves the olfactory tract, the bulb, or the nervous filaments, which are distributed to the nasal vault through the cribriform plate of the ethmoid. Disease of the middle cerebral artery near its origin from the circle of Willis may cause loss of smell in the corresponding nostril. This is probably through injury to the branches of the olfactory tract. Basilar fracture often destroys the nerve at the cribriform plate. In such cases the loss of smell is an important localizing fact. Localized meningitis, caries of bone, tumors, or abscesses may have the same effect. Hydrocephalus may seriously compress these nerves. It has been claimed that tumors situated in distant parts of the brain provoke neuritis in the olfactory tract similar to the optic neuritis usually associated with cerebrial neoplasms. In old age the olfactory bulb atrophies and the sense of smell diminishes. In hysteria smell may be entirely abolished. This may be a bilateral condition and exist practically alone, but is usually unilateral and associated with other sensory defects on the same side. Gowers mentions some cases in which the recognition of certain odors alone was lost. Overstimulation of the sense of smell will paralyze it, and the loss may be permanent. Ordinarily, a strong stimulation for three or four minutes so blunts the sense that it no longer recognizes the particular odor, but recovers itself in about a minute. Continued exposure to strong odors usually results in permanently diminishing this special sense. Oversensitiveness is occasionally noted in neurotics, and can be cultivated. The blind and those who taste tea or inspect certain articles of commerce gain great sensitiveness in this way. Finally, the olfactory bulb may be congenitally wanting.

The prognosis in loss of smell from disease of the olfactory nerve is usually bad. It depends on the cause in a given case. Catarrhal nasal conditions, fifth-nerve disturbance, and hysteria must be ruled out. The use of faradism is said to have done good in a few cases. It is applied to the mucous expanse over the turbinates, and is very painful. Snuffs containing strychnin or quinin may be tried. Oversensitiveness is controlled by morphia or cocaine, but their use is attended by the danger of setting up an incorrigible habit.

**Diseases of the Optic Nerve.**—The Visual Tract.—Only the infinitely short fibers between the layer of rods and cones and the retinal

<sup>1</sup> Zuckerkandl, "Ueber das Knochencentrum."

<sup>2</sup> "Arch. de Neurologie," 1885.

nerve-cells can properly be called peripheral optic nerves. The retro-ocular bundles that are named optic nerves by anatomists react to injury, as do other cerebral connecting tracts. Like them, if divided they never unite, while peripheral nerves unite readily under proper conditions.

The term optic nerve, with this understanding, will, however, be used as ordinarily accepted. The retina is made up of two lateral halves supplied from corresponding sides of the brain,—that is to say, the right half of each retina is in anatomical connection with the right cerebral hemisphere and the left half of each retina with the left hemisphere. At the macula lutea, or point of greatest visual activity, these halves overlap. This central part of the retina is thereby abundantly supplied from both hemispheres. As the bridge of the nose cuts off much light that would enter the pupil from that direction, the temporal or outer halves of the retina are rendered in part functionally inactive and their afferent fibers are less in number. The optic nerve in consequence contains many more fibers for the inner than for the outer halves of the eye-ground.

At the chiasm in man a partial decussation takes place. The larger number of fibers, those in relation with the inner or nasal halves of the retina, cross to the opposite side. Those from the temporal halves of the retina pass backward on the same side. At this point a number of fibers enter the gray matter in the floor of the third ventricle. Back of the chiasm the temporal fibers of the right eye are accompanied by the nasal fibers of the left eye, and they together make up the right optic tract. In other words, the right optic tract contains all the fibers going to the right halves of both eyeballs. It is, then, clear that while division of an optic nerve causes absolute blindness of one eye, division of one optic tract would produce half-blindness of the corresponding sides of both retinae. This would manifest itself in blindness in the opposite halves of the visual fields, *hemianopsia*, owing to the fact that oblique rays of light entering the pupil impinge on the opposed portion of the retina. A glance at figure 29 will make this clear. Owing to the fact that the macula has a double supply the hemianopsic field always shows an indentation at the fixing point. In such cases direct vision may not be impaired in acuity, so thoroughly is each macula supplied by both hemispheres. The dividing line between the half-fields in a hemianopsic eye is practically vertical, but may incline one way or the other to a slight extent in various individuals.

Injury dividing the chiasm longitudinally would cut off all the fibers to both nasal halves of the retina and produce double temporal hemianopsia, marked by blindness for all objects to the right for the right eye and all objects to the left for the left eye. Enlargement of the pituitary gland or pressure through the floor of the third ventricle may cause this result.

In extremely rare instances a bilateral blindness in the nasal fields is caused by symmetrical lesions in the optic nerves. This condition has been usually attributed to bilateral lesion of the outer portions of



the chiasm, but at this point the crossed and direct fibers are intermingled (see Fig. 29 A), and Shoemaker<sup>3</sup> insists that the process is due to neuritis invading the optic nerves symmetrically by way of the



Fig. 28.—Diagram of visual paths (after Slater)

filixus septi. These are quite uniform anatomical structures, as shown by Wilbrand and Sanger.

The optic tract passing backward encircles the crus cerebri and enters the geniculate bodies, the anterior corpus quadrigemini, and



Fig. 29 A.—Diagram of the optic chiasm, from Wilbrand and Sanger (after Hirschow)

the optic thalamus of the same side. From these ganglionic bodies fibers then pass outward and backward around the posterior horn of the lateral ventricle, to end in the cortex of the cuneus, the postero-

—N. Y. Med. Jour., Feb. 4, 1905.

internal portion of the occipital lobe. This portion of the occipital lobe represents, therefore, half-vision for each eye and is in relation with the

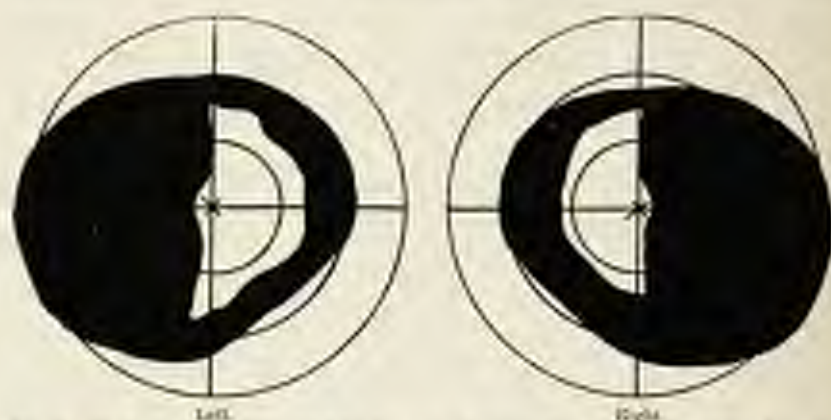


FIG. 30.—Blindness in both temporal fields in a case of meningitis due to injury of the chiasm by pituitary enlargement. The nasal fields are also contracted.

lateral halves of the retina on the same side of each eyeball. Any lesion that interrupts the visual pathway back of the chiasm, or destroys the visual centers in the cinereus, produces *lateral homonymous hemianopia*.

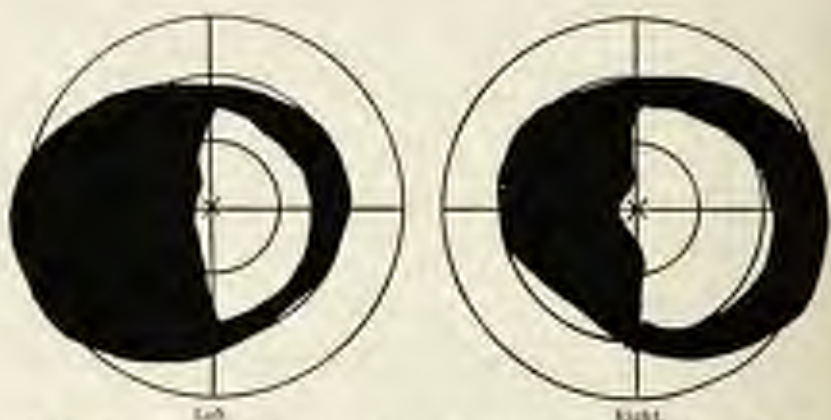


FIG. 31.—Homonymous lateral hemianopia from an injury to the right occipital apex. The right lateral fields are also contracted.

The cortical half-vision centers of the cinereus are in turn brought by conveying fibers into relation with higher centers for visual memories, probably situated in and about the angular gyri of the parietal lobes. In these higher visual centers both eyes are represented in each hemisphere. The parietal centers probably freely communicate through the callosal commissure. Injury to the left parietal region in right-handed persons produces loss of visual word-memories, or word-blindness, but does not necessarily cause hemianopia.



The fibers which supply the macula lutea of the retina occupy at the apex of the orbit the central portion of the optic nerve in close proximity to the central artery and vein. They then become superficial on the outer side of the nerve and proceed in this position to the back of the eye. Aneurysm of the central artery or axillary inflammation of the nerve about the artery may so involve them that blindness of the center of the field develops. This condition of central blindness, or *central*

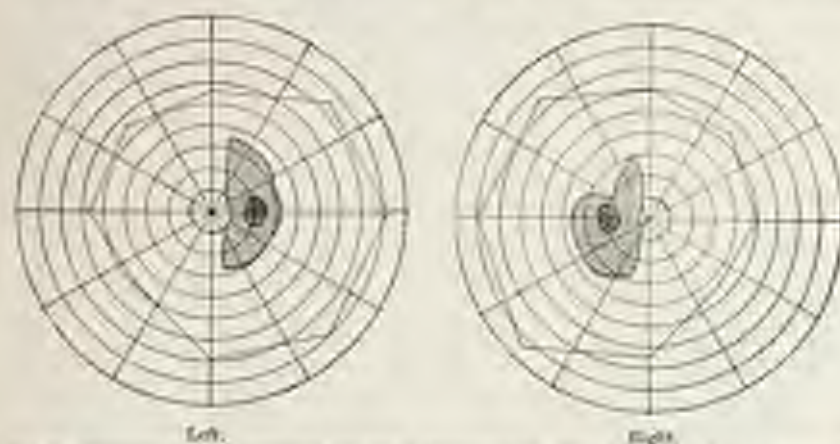


Fig. 22.—Scotomata in toxic amblyopia, consisting of increased size of blind spots, which are expressed by the darker shading.

*scotoma*, is also common in tobacco and alcoholic amblyopia. The poison seems to have the greatest effect on the most used and consequently most sensitive fibers or their related parts.

The loss of vision is sometimes limited to a quadrant of the field. The quadrant in the field is bounded practically by lines horizontal and vertical to the fixing point, which itself is spared. A case originally presenting hemianopia may eventually recover in part and a quadrant loss alone remain. It is probable that these quadrants are specially represented in the occipital cortex. Holmes and Lister<sup>1</sup> have determined with relative certainty that the cortical representation of the macula is at the apex of the occipital visual field, and that the upper quadrants of the retina are placed above the lower in their cortical relations. In organic hemianopia usually the seeing half of the field is also more or less peripherally reduced. The blind portion of the field may not be uniformly affected, some fractional vision remaining at various points. Rarely cases have been noted in which there was hemianopia for certain colors alone. In tobacco blindness the central scotoma, as a rule, varies for different colors.

In locating the lesion that causes hemianopia, the *hemianopic pupillary reaction* of Wernicke is of value. If the pupil responds when a narrow beam of light falls on the blind retina, the lesion is back of the geniculate bodies. The presence of this reaction indicates that the pupillary nerves are not involved. They accompany the optic tract as far as the

<sup>1</sup>"Brain," 1910, vol. xxxix, p. 34.

geniculate bodies. The test must be made with great care. The reaction has a positive significance when present, that its absence lacks. A double temporal blindness by itself is positive evidence of disease of the *chiasm*. When an *optic tract* is involved, the crus is almost invariably affected at the same time by the same lesion, so that paralytic body-symptoms on the same side as the blind field or opposite to the blind retinal half are present. Other cranial-nerve lesions are likely also to be present. Lesions of the *geniculate bodies*, the *corpora quadrigemina*, and the *optic thalami* almost invariably involve the internal capsule and produce paralysis in the body on the opposite side. A pure bilateral homonymous hemianopia may be produced by injury to the *cones*.

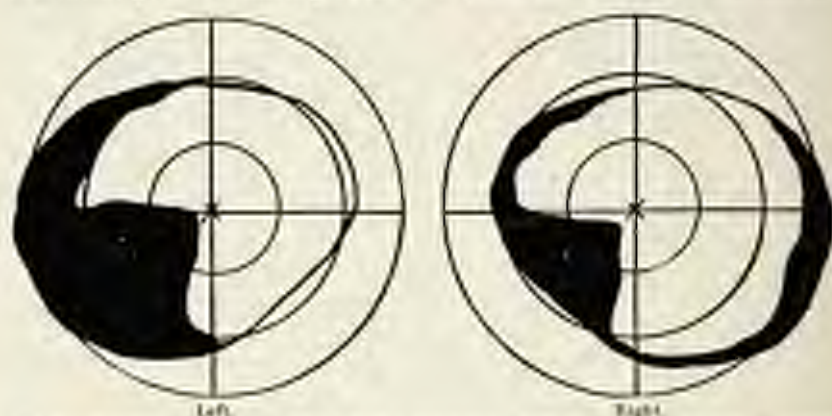


Fig. 33.—quadranitic loss of visual fields following meningitis. Some peripheral contraction also present in the right field.

A transient hemianopia occasionally is a symptom of migraine and lithemia. It is not impossible that hysteria may produce it, but concentric contraction of the field and changes in the color formula are usual in hysteria. In organic hemianopia the reductions of the field embrace form and colors equally.

The *optic nerve* is often injured by intra-orbital disease, such as tumor or aneurysm. It may be cut off at the foramen opticum by a basilar fracture, or involved by inflammation from caries of the sphenoid. An extension of inflammation down the sheath in meningitis is not rare.

The *chiasm* is injured most frequently by pituitary tumors, by syphilitic growths, and by pressure from the third ventricle in hydrocephalus. Gouty changes and interstitial hemorrhage have also been observed. Diseased carotids may press upon it.

The *optic tract* may be involved in basilar tumors or those on the internal aspect of the temporo-sphenoidal lobes. A patch of thickening in multiple sclerosis may affect it. The *intracerebral* path of the visual tract is often affected by tumors, hemorrhage, softening, and traumatism.

The *Papilla and Retina*.—Associated with or consequent upon many organic processes in the brain and spinal cord, the optic-nerve head and the retina are diseased. In two-thirds or more of the cases of cerebral tumors *papillitis*, usually double, is present at some period. It



presents ophthalmoscopically the *choked disc*. The onset, often sudden, may be insidious, and in some measure the rate of development is related to the activity of the new growth. While a long-standing growth in the brain may give rise to a sudden papillitis, a chronic process in the nerve-head is never associated with acute brain disease, except as a terminal condition or an accidental companion. Of much importance is the fact that a well-marked choked disc may not be attended by much loss of vision and no symptoms may call the patient's attention to its presence. In some cases temporary loss of vision, lasting from a few minutes to several hours, has been noted. Intense optic neuritis finally affects vision in all its modes. Acuity is diminished, the field is reduced for form and color, scotomata for both are frequent, and blindness may be induced. In most cases at some period the color fields will show variations and interweaving somewhat similar to those of hysteria. The papilla is swollen and infiltrated with a plastic material that obscures the outline and enlarges and elevates the disc. The organization of the infiltrate and consequent shrinking causes the final damage to the entering nerve-fibers. Papillitis may terminate in recovery, but its legitimate end is more or less atrophy of the optic nerve, with impairment of vision or complete blindness. In many cases of brain-tumor with optic neuritis the papillitis subsides on the removal of the tumor. Even opening the skull widely may cause a reduction of the papillitis in inoperable brain tumors and prevent blindness. In other instances improvement in the brain-lesion is attended by decreasing papillitis. The intensity of the papillitis, therefore, has some diagnostic and prognostic value.

Papillitis rarely results from brain-abscess, but in basilar meningitis a *papillo- or neuroretinitis* is extremely frequent. In this form the choking of the disc is less marked. Orbital disease is the usual cause of unilateral papillitis, but in rare cases a one-sided optic neuritis has been caused by tumor, generally on the same side of the brain. Wilder<sup>1</sup> notes that in ten cases where optic neuritis was distinctly greater in one eye than in the other, the tumor was on the side of the brain corresponding to the more intense inflammation. Marcus Gunn<sup>2</sup> found that the tumor and unilateral papillitis were located on the same side of the head in eighteen of twenty-four cases, and with greater uniformity in the tumors that were situated anteriorly. The localizing value of bilateral papillitis is practically negative, as it is common to tumors in all parts of the brain, but is particularly frequent with cerebellar growths and those situated in the brain-axis.

Optic neuritis also occurs in toxic conditions. It is sometimes found in severe anemia, often in albuminuria and in lead-poisoning, and after infectious fevers. In the albuminuric form the retinal expanse is commonly invaded, but sometimes the changes are practically confined to the disc.

Regarding the causation of papillitis, many theories have been advanced and rejected. In some cases it is clearly due to irritation descending the sheath of the optic nerve from intracranial inflammation. The idea that it is due to intracranial pressure or pressure within

<sup>1</sup> "Chicago Medical Recorder," June, 1894.    <sup>2</sup> "London Lancet," July, 1890.

the optic-nerve sheath in all cases has not been abandoned. Bondley and Cushing<sup>1</sup> contend that pressure producing distention of the optic nerve-sheath by cerebrospinal fluid is the sole cause, and support this contention with numerous clinical observations and animal experiments. In toxic cases it may represent the local action of the poison. Deutchmann<sup>2</sup> insists that it is due to pathogenic organisms which enter from without. Pressure is probably by far the most frequent cause, and the detection of a choked disc should always suggest abnormal intracranial pressure and the possibility of brain tumor.

**Atrophy** of the optic nerve may (1) follow papillitis and retinitis or chorioiditis; (2) it may result from injury or inflammation to the nerve-trunk; (3) it may be associated with sclerotic disease in the brain and spinal cord; (4) it may be due to diabetes, malaria, or syphilis; and (5) it may be of unknown causation.

The atrophy consecutive to papillitis is easily understood, and of the same nature are the retinitic and chorioiditic forms. Injury to the optic nerve naturally results in atrophic degeneration of the nerve-head. That form of optic atrophy found in about a tenth of the cases of locomotor ataxia, often present in parietic dementia, and not infrequent in multiple



Fig. 13.—Atrophic papillitis.



Fig. 14.—Atrophy of the optic nerve.

or disseminated sclerosis, has an importance quite its own. Atrophy is found in aneuritic idiosy and in the cerebellar form of family ataxia.

The symptoms and ophthalmoscopic pictures are tolerably uniform for the various forms. In the variety associated with tubes (the disc is often grayish, translucent, and shows the stippling of the lamina cribrosa. In the postpapillitic form the translucency and stippling are less marked. In aneuritic idiosy there is a peculiar bluish spot at the site of the macula about twice the size of the disc, presenting in its center a brownish-red spot strongly contrasting with its surrounding patch and resembling a central embolism or hemorrhage. At the same time the disc

<sup>1</sup> Jour. A. M. A., Jan. 30, 1909.

<sup>2</sup> Ueber Neuritis Optica, 1887.



is atrophic. In all forms of atrophy the disc is sharply outlined from the surrounding retina by its pallor and the vessels are diminished in size.

**Functional and Toxic Blindness.**—The peculiarities of hysterical blindness will be detailed in the description of that disease. In such cases the loss of vision may come on slowly or suddenly. It is usually unilateral, and most marked on the paralytic or anæsthetic side of the body, but the opposite eye is nearly always somewhat affected. The characteristics are contracted fields and inversion of the color formula. When apparently absolute, by using prisms or other suitable means, it can be demonstrated that the eye operates properly, but that ocular impressions are ordinarily neglected by the higher visual centers, where the disturbance must be located. In a very few such cases it is bilateral and complete, sometimes with dilated inactive pupils, in others with normally acting pupils.

Disease in branches of the fifth nerve is sometimes attended by partial blindness. This is marked by considerable eccentric contraction of the field and some loss of acuity of vision. It is principally associated with disturbance in the dental branches, particularly those to the molar teeth.

Acute *swainia*, the result of excessive hemorrhage, sometimes induces blindness that may be complete and permanent.

A number of general *toxic states*, such as uremia and syphilis, and those due to quinin and lead, may cause amblyopia. Uremic blindness is often of sudden onset, but may be preceded by dimness or haziness of vision. The fundus in such cases usually presents albuminuric retinitis and Bright's disease is present. Temporary or recurrent blindness in syphilitic involvement of the brain is sometimes a valuable early diagnostic symptom of that condition and also of brain-tumor. Early correction of the toxic state in the various poisonings usually promptly results in a return of vision, but the persistence of toxæmia may induce permanent blindness.

Excepting in the albuminuric cases, the fundus at first ordinarily presents nothing abnormal. Sometimes pupillary symptoms are absent; sometimes there is dilatation and immobility. The difference seems to be due to the essential involvement of the cortical cells in the first case, and of the retinal cells, or of both, in the second.

No attempt is made in the foregoing to exhaustively treat of the diseases of the visual tract and retina. The involvement of the second cranial nerve is commonly only a part of a neurological case, but has a symptomatic value of great importance. From this standpoint the various features that relate to diseases of the nervous system have been grouped.

#### CHAPTER IV.

#### DISEASES OF THE OCULAR NERVES—THE THIRD, FOURTH, AND SIXTH CRANIAL PAIRS.

**Anatomical Considerations.**—The muscles of the eye are supplied by the third, fourth, and sixth cranial pairs and the sympathetic. The third, fourth, and sixth nerves arise from collections of cells ranged from

before backward, under the posterior portion of the middle ventricle, the aqueduct of Sylvius, and the anterior angle of the fourth ventricle.

They have in common the function of controlling the movements of the eyeballs. In addition, the third nerve governs contraction of the iris and the ciliary muscle, and elevation of the upper eyelid. The sympathetic, through the optic ganglion, dilates the pupil. Starting from before backward in the nuclei of the third nerve, we find represented (1) ciliary and then (2) pupillary contractions. The centers for the extrinsic ocular muscles follow presumably in the following order: The internal rectus, the superior rectus, the elevator of the eyelid, the inferior rectus, and the inferior oblique. The fourth nerve is distributed solely to the superior oblique, and its center is placed close behind those for the third nerve. At a little distance caudad under the floor of the fourth ventricle is the center for the sixth nerve, which controls the external rectus. All these centers are in close functional and anatomical relationship, and can reasonably be considered as a single mechanism, made up of three portions. The first is the iridociliary, the second embraces the centers for

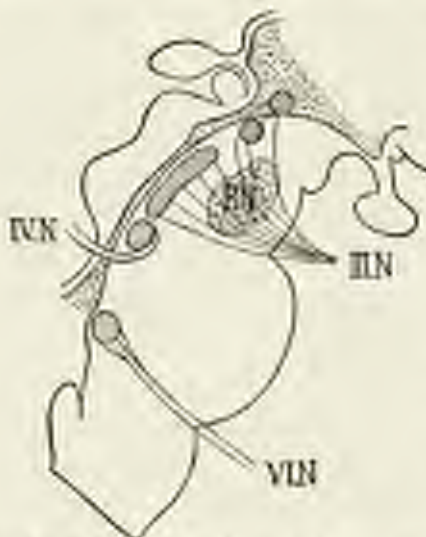


Fig. 36.—Diagrammatic longitudinal section of the midbrain, showing the positions of the nucleus tractus for the ocular muscles (after Sherrington).

the muscles of convergence, and the third comprises the nucleus for the sixth nerve, which controls the external rectus, the only divergent ocular muscle. Ocular movements are also represented in the frontal motor cortex. The nucleus centers are connected with the higher levels through the motor radiations and the internal capsule. The muscles governed by the third nerve are also brought into relation with the orbicular muscles of the lids by nuclear connection with the facial nerve. The accommodation and light reflex are thereby correlated to the act of winking and the position of the palpebral curtains.

Based upon the studies of Schaefer, Unverricht, Duailley, Mink, and his own experi-

ments, Rosta<sup>1</sup> asserts that the oculomotor apparatus has a double cortical representation; first an anterior one at the foot of the second frontal convolution, and, second, a posterior center in the occipital region. Von Bechterew<sup>2</sup> also contends that irritation of the anterior margin of the occipital lobe in the dog produces narrowing of the pupil and increase of accommodative effort.

The various ocular muscles serve to move the globe in the orbit in the directions indicated by their names; but the superior and inferior recti, owing to the oblique direction from the apex of the orbit to their

<sup>1</sup> "Arch. de Neurol.," Sept., 1890.

<sup>2</sup> "Neurolog. Centralblatt," May, 1890.



insertion, also draw the eyeball toward the nose and rotate it inward. This is counteracted by the oblique muscles and the external rectus, but the oblique muscles also act in convergence. Convergence of the eyes, necessary for all close vision, is thus much better provided for than the opposite action. Voluntary divergent squint is impossible.

The motor nerves of the eye may be diseased (1) at their nuclear origin, (2) in their intracerebral course, (3) in their intracranial course from apparent origin to their cranial outlets, and (4) within the orbit. Four groups of symptoms are produced: (1) Pupillary variations, (2) disturbance of accommodation, (3) muscular incompetence and squint, (4) double vision. It is by the study of these symptoms, their mutual combinations, and the association or absence of other cerebral indications that a distinctive and localizing diagnosis is possible. We will first consider the individual nerves.

**Ocular Palsies.**—Complete division of the *third nerve* paralyzes all the extrinsic muscles but the external rectus and the superior oblique. The eye, in consequence, can be turned strongly to the outer canthus, and eventually becomes fixed in that position. The eyelid droops in complete ptosis, which the patient tries to remedy by the action of the frontalis. The pupil is widely dilated by the unopposed action of the sympathetic, and does not react for light or accommodation. The eye, unless strongly myopic, is incapable of near vision through the loss of accommodation due to paralysis of the ciliary muscle. There is double vision excepting when the sound eye is made to correspond with the direction of the one paralyzed.

Division of the *fourth nerve* allows the eyeball at the top to slightly rotate outward on the anteroposterior axis, which corresponds practically to the line of direct forward vision. When fixed in this position by the action of the inferior oblique and the inferior rectus, which act together, the rotation can sometimes be observed if carefully looked for, but the index to this paralysis is in the diplopia, to be studied later. In



Fig. 12.—Paralysis of the left third cranial nerve. 1, 2, 3, Different degrees of deviation of eyeball in various positions. 1 shows partial ptosis and outward deviation of affected eye.

attempts at downward convergent vision the inward rotation of the eyeball fails to take place.

When the *sixth nerve* is divided, the eye can not be turned outward from a line marking direct forward vision, and later the eye becomes

fixed in a strong inward squint that may carry the pupil to the internal canthus. The diplopia is marked.

Destructive injury to the sympathetic produces great narrowing of the pupil, as the pupillary sphincter, innervated by the third nerve, escapes. At the same time the eyeball becomes prominent or slightly exophthalmic by reason of the paralysis of Müller's muscles and the retro-ocular turgescence. The pupil does not react for light or for pain.

**Diagnosis of Ocular Palsies.**—When the lesion is complete and has existed some time, difficulty in deciding which extrinsic muscle is at fault is not very great. In partial lesions and when the condition is vacillating or slight in degree, a careful examination is required. We have two important indications—namely, the *habitual position of the head* and the *diplopia*. When an ocular muscle is weakened, the patient unconsciously and constantly so carries the head that the least possible work is required of the paretic muscle. For instance, if the right external rectus is involved, the patient will turn the head to the right, thereby relaxing the injured muscle. The rule as laid down by Landolt<sup>1</sup> is that *the direction of the head corresponds in every way to the physiological action of the paralyzed muscle*.

If the extended lines of the visual axes for both eyes do not attain the same fixed point, diplopia results. The sound eye sees the fixed object clearly and the mind refers the image to the proper position in space. The divergent or convergent eye sees the fixed object indistinctly, as the image does not fall on the sensitive spot of the retina and the mind projects it to a wrong position in space,—namely, to the position by experience associated with the particular part of the retina in operation. In diplopia of long standing the mind learns to neglect the weaker, false image, and the patient may be unaware of his double vision. The eye also usually becomes more and more divergent, presenting the comparatively insensitive retinal periphery to the fixed object, and this assists in the mental neglect of its image. When the muscle weakness is very slight, diplopia only occurs on quick movements of the eyes or in extreme range of the eye in the direction of the affected muscle's action, and then it may be but momentary, the muscle under the stimulus of attention gradually drawing the eye into line. This is often accomplished in a jerky manner. The eyeball presents a number of oscillations which may terminate in the proper position, or the globe may swing back to the abnormal position, the muscle being quite exhausted. The double vision and the faulty projection at first give rise to pronounced ocular vertigo.

In examining a case of diplopia the first question is whether it is monocular. Closing one eye prevents double vision unless it is confined to the open eye, in which case, if not due to defective media or faulty curvatures, it is almost surely hysterical. Place the patient in a good light, have him hold the head motionless, and let him follow with both eyes the point of your finger through a circular range about a foot from his face. At some angle the two eyes will fail to maintain parallelism, or jerky movements of one eye may be noticed. The point is to determine which is the affected eye. Cut off the patient's line of sight from one eye with a card held at such a distance from it that its motion can still be ob-

<sup>1</sup>"Brit. Med. Jour.," Sept. 15, 1894.



served, and have the patient fix both eyes upon your finger in the direction toward which conjugate movements failed. If the sound eye be covered, it will overact and be turned farther to the side than is required. This overaction, or *secondary deviation*, is due to the fact that the stimulation necessary to draw the affected eye in the direction of the weak muscle is more than sufficient for the corresponding sound muscle. If you have covered the weak eye in the same way it will not move outward far enough when the finger is fixed by the sound eye, and for a similar reason. The direction of failure in the affected eye, therefore, points to the weakened muscle. In marked squint of this character the affected eye often presents a much wider range of motion when the sound eye is covered than when binocular vision is attempted. Apparently the volitional attempt is stronger when the muscle is not disturbed



Fig. 26.



Fig. 27.

Fig. 26. Convergent squint. The ray of light, *a c*, falls to the right of the sensitive spot, *w*, of the uncovered left eye, *L*. Such stimulus has always been recognized by the mind as emanating from objects to the left, and not restoring the disordered position of the ocular globe a false image, *a'*, is projected an additional distance to the left, measuring from *c* at the opening sensitive spot, in the line *a' c'*.

Fig. 27. Divergent squint. The ray of light, *a c*, falls to the left of the macula, *w*, of the outwardly turned right eye, *R*. Such stimulus has always been associated with objects to the right, and the false image, *a'*, is projected to the right, *c'*, setting out the sensitive spot in the line.

by two images. Overaction of the sound eye, however, occurs, as in the card-test.

The *diplopia test* is made as follows: Place the patient facing a candle or bright object, such as a narrow strip of white paper the size of a lead-pencil, placed on a dark background at a distance of, say, twenty feet. Cover one eye with a red glass. If there is double vision, the red image corresponds to the covered eye. If the red image and the covered eye are on the same side, the diplopia is *simple*; if on opposite sides, the diplopia is *crossed*. When the eyes converge, the diplopia is simple; when the eyes diverge, the diplopia is crossed. Gowers' rule is: "When the visual lines, the prolonged visual axes, cross, the diplopia is not

crossed." If, now, the fixing object remaining stationary, the head be turned one way and another,—in some given direction the images will separate and in the opposite direction approach and perhaps fuse. Landolt<sup>1</sup> lays down this rule: "*The affected eye is that to the direction of whose image the diplopia increases.*" For instance, in simple diplopia, the right image corresponds to the right eye. If the diplopia increases on looking to the right, the right eye is affected. In crossed diplopia the left image corresponds to the right eye. If the images separate on looking to the left, the right eye is at fault because its image corresponds to the direction of the increased diplopia. The same thing is true in vertical diplopia.

He also lays down this rule: "*The paralyzed muscle is the one which would give to the eye the direction of the false image.*" The false image, of course, belongs to the affected eye. For instance, in simple diplopia with the right eye affected, it is the external rectus that is at fault, as it is this muscle which would, if competent, direct the eye to the position of the false image at the right of the true image. Involvement of the oblique is similarly shown. If the patient indicates that the image of the right eye, in simple diplopia, is to the right and its upper end leans to the left, we know, by the application of this rule, that the right superior oblique is deficient. An opposite condition would point to the inferior oblique and inferior rectus, which separate together.

The location of the lesion determines the distribution of symptoms and through them is clinically deciphered. A lesion involving the nuclei under the aqueduct of Sylvius is almost invariably attended by bilateral symmetrical eye conditions. This is due to the anatomical proximity of the centers on each side of the middle line and to their physiological relations. A lesion which destroys accommodation and pupillary contractions affects the *most forward group* of nuclei. If the middle group is involved, all the extrinsic muscles of the eyes are disabled, even the external recti, as this group has a controlling influence over the nucleus of the sixth nerve. If the lesion fall upon the *most backward group*, double convergent squint is produced. In the same way, if some one or several of the conjugate movements of the eyes be lost, or a double ptosis is present, the lesion is nuclear unless movements of the head and trunk are lost at the same time. In such case the lesion is probably above the nucleus in the internal capsule or in the cortex.

The three ocular nerves leave their grouped centers by widely diverging roots, and may be cut off separately and unilaterally by cerebral conditions, such as hemorrhage, inflammation, or new growths. Such lesions invariably implicate other structures, and give rise to numerous symptoms other than those referable to the ocular nerves. For instance, a lesion in the peduncle in the region of the red nucleus would disturb the motor tracts in the crus and produce paralysis on the opposite side of the body, with paralysis of the third nerve, ptosis, etc., on the same side as the lesion. This, by Charcot, is designated the symptom group of Weber (fig. 26).



In basilar inflammations and injuries, and particularly in syphilitic meningitis, the ocular nerves are likely to be injured. The sixth, from its long and exposed course, is especially prone to injury and is the most frequently diseased nerve of this group. Lesions in this position are usually unilateral, and if bilateral differ, as a rule, in degree on the two sides. Here injury to the third nerve necessarily involves all its branches and functions. A lesion which could select from the nerve-trunk the papillary fibres, for instance, is inconceivable. Diffuse cerebral symptoms, such as headache, vertigo, nausea, stupor, convulsions, and optic neuritis, are also commonly present. At the apex of the orbit all the ocular nerves, with the ophthalmic division of the fifth and the optic nerve, are closely approximated. Injury or disease at this point would, therefore, determine complete internal and external ophthalmoplegia, impair vision, and give rise to sensory disturbances in the distribution of the supra-orbital nerve. Pressure at the same time on the return orbital circulation congests the retinal veins and produces some exophthalmos. After the nerves have separated in the orbit, any one or more of them may be injured by transection or local disease. Their branches may be singly selected. The diagnosis depends upon the muscles involved, and the anatomical relations of the nerves and their branches within the orbit.

**Causes of Ocular Palsies.**—The conditions which give rise to palsies of the ocular muscles are very numerous. In the orbit transection and new growths are frequently causal of the loss of ocular movements. A blow over the eye is sometimes followed by paralysis of the levator palpebre superioris. Exposures to cold and the rheumatic states are sometimes followed by an ophthalmoplegia, which is probably due to a peripheral neuritis. This form may be present in multiple neuritis from alcohol, and probably from other poisons, and may involve few or many of the muscles. Orbital growths usually are marked by exophthalmos. Cervical, maxillary, and cranial tumors may invade the orbit and there produce these local symptoms.

In their basilar course the motor nerves of the eye are frequently injured by meningitis, and especially by syphilitic disease. Basilar fracture, carotid aneurysm, or the pressure of neighboring intracranial growths may also implicate them in this locality.

At the onset multiple sclerosis, bulbar palsy, polioencephalitis superior, and locomotor ataxia frequently cause ocular palsies. A temporary ptosis or diplopia is often an early symptom of tabes, and should always arouse suspicion of it or of other syphilitic diseases. The exact mechanism and location of the tabetic disturbance which produces the Argyll-Robertson phenomenon is not understood, but presumably the anterior cell-group, controlling pupillary and ciliary contractions, is involved. In myasthenia gravis various ocular deviations and a marked tendency to ptosis are encountered.

A peculiar periodically recurring or relapsing palsy of the third nerve is sometimes encountered. It is more frequent in females, involves only one eye, lasts from one to six months, and returns at regular intervals, often during the entire life of the patient. Its pathology is not known.

but the nerve-trunk has several times been found to be diseased. Traces of this palsy, in some cases, can be detected during the intervals of practical freedom from the trouble. It has been attributed to hysteria in certain instances, and is frequently associated with migraine, the so-called *ophthalmoepileptic migraine*.

*Lesions of the cortex or of the internal capsule producing hemiplegia* are often attended by conjugate deviation of the head and eyes toward the sound side of the body, but if the paralyzed side present convulsions due to cerebral irritation, the eyes are then spasmodically drawn to that side. A lesion lower down in the pontine region would be marked by a crossed deviation, the eyes looking to the paralyzed side of the body, and in convulsions turning to the sound side of the body.

**Ocular Muscle Spasms.**—The ocular muscles are usually associated with the other muscles in generalized convulsions, the eyes turning to the side most vigorously affected. Individual muscles alone are affected in very rare instances. The levator or a rectus may thus be spasmodically involved for years. Sometimes the ocular muscles participate in facial ties and spasmodic torticollis. In hysteria the eyes are frequently rolled upward and outward, or strongly converged during convulsive manifestations of the disease, and these ocular positions may remain for long periods of time after the convulsion has subsided. A more common eye condition in hysteria is one of spurious double vision. It is really a spasm of the ciliary muscles. The patient apparently tries to overcome it by raising the brows with the frontalis. An attempt to raise the lids with the finger at once develops resistance, which in paralytic ptosis is never present.

**Nystagmus** consists in rapid rhythmical involuntary oscillations of both eyeballs, due to spasmodic action of the ocular muscles. When both eyes are affected, the movements are similar and synchronous. The spasmodic movements are commonly horizontal, less frequently rotary, and only rarely vertical. When the eye vibrates in one plane, it ordinarily moves outward quickly and more slowly returns. The oscillations vary in number from fifty to two hundred or more a minute, and in extent from one to ten millimeters. In uremic coma the eyes will sometimes be noticed in a rhythmical movement which carries them through their widest lateral range about ten times a minute. Nystagmus may be constant or only provoked by calling forth a particular movement of the eyes, as by directing them upward or outward. This latter must not be confused with the jerky, arrhythmical, and momentary movements of weakened muscles. Bérard claims that in about one-half of all normal persons strong lateral deviation of the eyes is attended by slight nystagmoid movements, but these are about equal for all extreme angles of vision. In the nystagmus of labyrinthine disease the movements are of unequal rapidity and are intensified in looking in the direction of the quicker movement. In organic disease of brain and cerebellum the nystagmic movements are persistent and practically of equal rapidity in each direction.

The causes and significance of nystagmus are often obscure. It may be acquired as the sequel of any condition which greatly impairs vision. Corneal and lenticular opacities, choroiditis, and retinitis may precede it.



especially if occurring in childhood. Albinos frequently present nystagmus. Miners who work in cramped positions with poor illumination acquire it, probably as a fatigue neurosis through the overstrain of certain eye-muscles in attempting to keep the work in view. In them it is often only present when the particular attitude which their work requires is assumed.

Nystagmus is present incidentally or regularly in a long list of nervous diseases which have neither location nor pathological anatomy in common. It is almost a cardinal symptom in insular sclerosis and in hereditary ataxia of the Friedreich type. It is common in cerebellar tumor and tumors involving the corpora quadrigemina and optic thalami. Occasionally it attends sinus thrombosis, meningitis, meningeal hemorrhage, and variously seated cerebral hemorrhages, softenings, and tumors. Hysteria has also furnished rare cases of persistent nystagmic ocular spasm. It is closely associated with auditory diseases affecting the labyrinth and often attended by vertigo and tinnitus aurium. (See Diseases of the Eighth Cranial Nerve, Chap. VII.)

The treatment of ocular palsies depends upon the causal condition and is often most unsatisfactory. New growths and traumata are surgical conditions for the most part. Syphilitic palsies may readily yield to treatment, whether due to local or central involvement, but only too often recur, yield again, and finally become permanent. The neuritic form is treated as a part of the general intoxication usually at the bottom of the disease. A persisting ocular deviation is often benefited by a tenotomy. Muscle cutting should always be preceded by exercise of the muscles with prisms, which sometimes is helpful, and by general treatment. Nystagmus, excepting in the unusual cases where it is due to meningeal and sinus diseases, or other curable lesion, is practically unyielding to all measures. Miners' nystagmus usually ceases when the occupation is changed.

## CHAPTER V.

## DISEASES OF THE TRIFACIAL NERVE.

**Anatomical Considerations.**—The fifth nerves represent the sensory portions of all the motor cranial nerves. Their sensory distribution embraces most of the skin of the head and face, all their mucous membrane-lined cavities, and the cerebral meninges in part. The exact limits of this sensory field have been worked out by Cushing in a number of cases subjected to extirpation of the ganglion of Gasser, and are shown in Fig. 39 A. In addition, through at least the chorda tympani, the fifth subserves the special sense of taste. Its small



FIG. 39 A.—Diagram showing the normal (average) field of postoperative sensory anesthesia. The shaded area, including hair and anterior wall of nostrils, represents distribution to facial (hair roller diameter) branch. The dotted strip gives the impression of touch or pressure to pain (thumb) branch, with few if any actual pain points (Cushing, in Bull. Johns Hopkins Hosp., July-Aug., 1904).

motor root innervates the mandibular muscles of mastication. The nuclear origin of the fifth nerve in the medulla is correspondingly extensive. The smaller motor nucleus is situated under the floor of the fourth ventricle near its lateral angle, with an upward extension as high as the corpora quadrigemina. Outside of this is the larger sensory nucleus, which is connected continuously with nuclear gray matter as low as the fourth cervical spinal segment. These centers are brought into relation with the cerebellum and with the cerebral cortex by special radiations. For the motor portion the cortical centers are at the foot of the anterior central convolution. The sensory cortical representation is not clearly known, but is probably in the same territory





**Cortical Diseases of the Fifth Nerve.**—Lesions of the portion of the motor cortex associated with the fifth nerve produce spasm or paralysis according as the lesion is irritative or destructive. The involved muscles are the pterygoids, the temporal, and the masseter. In generalized convulsions they are seldomly involved. Very rarely they are the sole seat of motor disturbance presenting a satisfactory spasm or paralysis. The spasm may be tonic, as in trismus, or repeated at varying intervals. The teeth are tightly clenched, and the masseters and temporals stand out firm and hard. In the clonic variety, as in the clattering of a chill, the lower jaw is sharply moved laterally or vertically. Vigorous grinding of the teeth may be present. This is sometimes seen in the late stages of parietal dementia and in other wide-spread organic brain disease. Single or repeated spasms may have their origin in local disease of the jaws, such as periodontitis or tooth-ache, and are then reflexly produced.

Masticatory paralysis of cortical origin is extremely rare. The cortical lesions have almost invariably been found to be bilateral, but



Fig. 11.—Cortical disease of the fifth cranial nerve in a case of tumor, showing loss of sensory and motor facial activity in this patient.

Hirt<sup>1</sup> has reported a case of complete mandibular palsy due to a left-sided lesion at the foot of the ascending frontal convolution.

**Nuclear disease of the fifth nerve** is usually a part of a group of bulbar symptoms. Masticatory paralysis arising from this source is extremely rare. Progressive bulbar palsy and posterior tumors may cause it, but other cranial nerves are almost invariably affected at the same time. Multiple sclerosis and tubes may and often do affect the fifth nucleus, causing bilateral sensory and motor symptoms in the area of its distribution.

**Peripheral intracranial affections** of the trigeminus may involve the stem, the Gasserian ganglion, or the three branches of the nerve at their exits from the skull. The differential diagnosis as to location may be impossible if adjoining nerves or structures are uninjured or present

<sup>1</sup> *Brain*, 111, 1908, 1887.



no indications. A description of the symptoms arising from injury to the trunk will therefore apply to disease of the Gasserian ganglion or of the three branches at their cranial exits. Growths and inflammatory processes are the usual causes of this form of trifacial disease.

The motor symptoms are those of paresis or paralysis of the mandibular muscles. The jaws can not be closed or can not be firmly held together when closed. The combined strength of the jaw-muscles is immense, and considerable impairment may go unnoticed. In complete bilateral paralysis the jaw droops, but can at first be raised by the action of the facial muscles, especially the buccinators and orbicularis oris. If the palsy is one-sided, the jaw may still be raised by the unilateral action of the uninjured side, but the bite is feeble. Attempts to bring the paralyzed pterygoids into play fail to produce grinding movements, so that the jaw can not be forcibly advanced from the impaired side or thrust in the opposite direction. In long-standing cases contracture in the muscles which depress the jaw may permanently hold the mouth open.

Interference with sensation may be partial or complete. In the latter case all parts of the face, head, nasal fossa, conjunctiva, mouth, and tongue supplied by the fifth nerve are insensitive, and taste is abolished on that side of the tongue and oral cavity. The sneezing reflex is abolished on the anesthetic side, as well as the gagging reflex caused by irritating the soft palate. Instruments may be passed into and through the nasal chambers without eliciting sensation, and ammonia fumes, etc., no longer cause irritation. Profound pricking, tingling, and burning usually precede the anesthesia. Frequently, when the loss of sensation is pronounced, so that the patient no longer feels a pin-prick, complaints are made of pain and burning in the anesthetic area,—*anesthesia dolorosa*. In one case observed by the writer, while general sensation was abolished in all its modes and tones, muscular sense remained. A touch or prick was not perceived, but the slightest motion communicated to any facial muscle was instantly recognized, apparently through the uninjured seventh nerve. *Trophic disturbances* are the rule, but, ordinarily, they are slight in degree. The insensitive conjunctiva and cornea are easily irritated and prone to ulceration that may reach a destructive grade. The nasal and lacrimal secretions are defective and the mucous membrane dry. In the nose this dryness impairs the sense of smell. The insensitive side of the tongue is thickly furred, due partly, but not wholly, to the fact that food is only chewed on the sound side. The salivary secretion may be greatly diminished. Herpetic eruptions in the cutaneous distribution are frequent, and when the ganglion or branches are diseased and the conjunctiva is involved, constitute a serious feature, as *ophthalmia* and complete loss of the eye may ensue.

Disease of the trifacial branches is extremely common. Their course through bony channels, serving to protect them admirably under ordinary conditions, exposes them to pressure from inflammatory states, to injury by crushing blows, and to laceration from fractures involving the cranial and facial bones. Their proximity to the nasal, buccal, and pharyngeal cavities, always containing the potential factors of infection,

is a local disadvantage. Finally, they are distributed to the most exposed portion, of the cutaneous expanse, where, thinly covered, they rest upon the unyielding structures.

They are very often the seat of *neuralgic pruritus*, which will be more particularly considered in the section on Symptomatic Disorders of the Nervous System, Part VIII. It is probable, however, that a nerve, the seat of long-continued neuralgic pain, symptomatic, perhaps, of a general blood state,—as malaria, for instance,—may eventually become histologically changed and organically diseased. A *neuritis* may be thus established which is usually marked by sharply defined anatomical areas of hypersensitiveness or anesthesia. Dystrophic changes in the dermal structures, such as sculiness, herpes, and falling or discoloration of the hair of the eyebrow and beard, are frequently encountered. The glands supplied by the given nerve over- or underact as the condition in the nerve is irritative or destructive. Neuritis may also be set up by extension from a neighboring inflammation in the orbit, antrum, or jaws. The dental branches are particularly liable to infection, injury, and irritation, which in the case of molar teeth is not infrequently the cause of pain referred to other branches of the trifacial than the one immediately concerned.

Injury to the lingual branch of the third division of the fifth nerve, if it occurs below the junction of the chorda tympani, produces loss of taste on the anterior portion of the tongue on the same side, in addition to the loss of general sensation and trophic disturbance in its anatomical area of distribution.

The motor fibres of the trifacial, being entirely confined to the third division, suffer with it. Paresis or paralysis of the muscles of mastication on the same side follows. The prominence and hardness of the masseter and temporal do not take place when the patient attempts to clench the teeth, and deviation of the chin to the opposite side can be but feebly produced or is lacking. Destruction of the *motor root*, either above or below the ganglion or at the bulbar nucleus, gives rise to muscular atrophy in the muscles of mastication, and to the reaction of degeneration upon their electrical stimulation. A cortical lesion causes no such degeneration.



## CHAPTER VI. DISEASES OF THE FACIAL NERVE.

**Anatomical Considerations.**—The seventh cranial nerve has its cortical origin or representation in the lower Rolandic region. The nuclear center is situated under the floor of the fourth ventricle, to the inner side of the ascending root of the fifth nerve. The pathways between medulla and cortex decussate in the median raphe (Eldinger). From the nuclear cells the nerve passes close to the nucleus of the sixth nerve, and, descending thence through the pons, emerges in the furrow between the pons and medulla outside the sixth nerve, closely accompanied by the eighth or auditory nerve, with which it proceeds directly to the internal auditory meatus. Its relation to the sixth nucleus and its proximity to the sixth nerve on emergence explain the frequent association of these nerves in disease. The parallel course of the auditory and facial from medulla to meatus explains why meningeal and basilar conditions of necessity affect them both at the same time.

The nucleus of the seventh receives fibers from the oculomotor nuclei above, which are destined to the orbicular muscle of the eyelids. By this mechanism the functions of wrinkling, accommodation, and ocular movements are associated. Fibers from the hypoglossal nucleus below also pass to the nuclear centers of the seventh, and are eventually distributed to the orbicular muscle of the mouth, correlating the labial and lingual movements necessary in phonation, mastication, and other buccal processes.

The decussation of the seventh explains the phenomena of crossed or alternate paralysis of the face and limbs. A lesion in the pons above the decussation involves at once the seventh nerve and the pyramidal tract for the opposite side of the body, but below the facial crossing and above the pyramidal decussation a lesion involves the face on the same side and the limbs on the opposite side. Such a lesion must involve the lower third of the pons, approximately the portion below the superficial origin of the fifth pair.

After entering the internal auditory meatus the seventh nerve bends somewhat sharply, and presents a gangliform swelling, which receives the large superficial petrosal from the Vidian nerve, probably containing the taste-fibers from the second branch of the fifth nerve by way of the sphenopalatine ganglion. The taste-fibers leave the facial nerve in the form of the chorda tympani after it has almost completely traversed the Fallopian canal,



FIG. 42.—Diagram showing the course of facial and auditory fibers, and the mechanism of crossed syndromes. A, Auditory nucleus associated syndromes; F, lesion causing crossed paralysis of the face on one side and the limbs on the other.

and, passing up through the tympanum, finally reach the anterior portion of the tongue with the lingual branch of the fifth. Within the Fallopian canal the facial gives off from within outward, first, near the ganglion of the knee, above mentioned, a motor branch to the tympanic plexus; second, a motor branch to the stapedius muscle; and, third, the chorda accom-

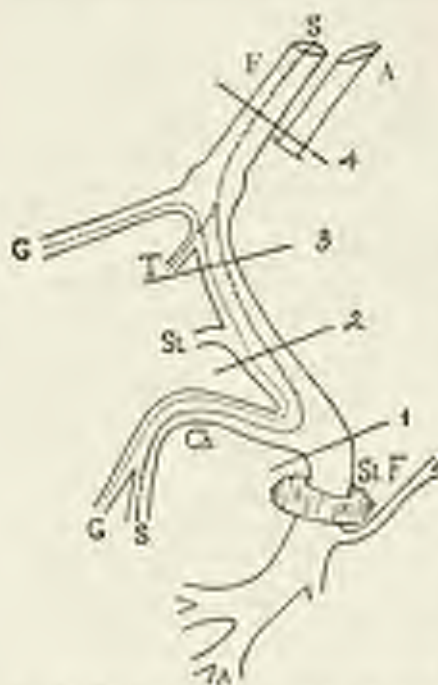


Fig. 45.—Diagram of facial nerve passing through the stylomastoid foramen. F, Facial; S, sensory; A, auditory; T, tympanic; St, stapedius; Ca, chorda tympani; G, geniculate ganglion; Sl, stylomastoid foramen; 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, branches.

panied by a secretory branch to the salivary glands. The facial nerve, therefore, within the aqueduct contains (1) motor filaments for the facial muscles, (2) filaments of the special sense of taste for the anterior two-thirds of the tongue, (3) motor filaments for the internal ear, and (4) secretory fibers for the salivary glands. In addition there are a few filaments subserving common sensation for the external ear, derived from the fifth nerve, which pass with the facial to its exit.

After leaving the stylomastoid foramen the facial gives off (1) the sensory branches above referred to, (2) branches to the external auricular muscles, and (3) branches to the posterior portion of the occipitofrontalis. The trunk in the substance of the parotid then divides into (4) widely spread branches supplying motion to all the muscles of

the face, to the platysma, the stylohyoid, and the posterior belly of the digastric.

Practically, the seventh nerve may be considered one of pure motion. It furnishes the impulse supply to the muscles of the face. The sensory and secretory filaments merely join it during its course and leave it before it reaches its periphery. When it is diseased the major symptoms are motor and consist of increased or decreased activity, giving rise to facial spasm or facial palsy as the condition is one of irritation or deficit. The muscles also waste. The addition of sensory and secretory disturbances enables us to locate the lesion with more or less exactness.

**Spasmodic Affections of the Facial Nerve.**—**Facial Spasm.**—Irritative lesions in the Rolandic face-centers may set up grinning spasms on the opposite side, usually of an irregular, clonic sort, which are not suggestive of any intention. Such a spasm may be the initial feature of a Jacksonian fit. Grinning is the feature of all severe epileptic attacks that most impresses onlookers. In limited cortical lesions in the face-centers the spasm may be confined to a per-



tion of the face, as to the angle of the mouth or to the eyelids and brow. These cortical spasms are usually attended by mental symptoms, such as an aura, confusion, or unconsciousness, and are practically independent of circumstances and surroundings. Feelings of apprehension and a distressed appearance commonly attend them. Their frequency is variable. Often they occur singly, at long and irregular intervals. An explosion, a sort of status, may take place so that the face is convulsed for many minutes or even hours, and then returns to a normal quietude, perhaps marked by a slight paresis or even by a distinct weakness, which is important and should always be sought.

The irritative lesion may be subcortical and produce the same conditions. At the nuclear level it would almost certainly involve other cranial nerves. Pontine and basilar growths have in rare instances been the cause of such facial spasms, and inflammatory invasion of the nerve-trunk is usually marked by twitchings in the muscles which subsequently present palsy. These twitchings often reappear with regeneration. All the above conditions produce a pure spasm.

Spasm in the facial distribution of a most inveterate character not associated with any pathological element of an anatomical nature is rather frequent. Any peripheral irritation in the nasopharynx or mouth may apparently cause it, and correction of such local factor lead to its cure. Usually some point can be found pressure upon which inhibits the spasm temporarily. Neurotomy, nerve stretching, alcoholic injections, and the use of the various nerve sedatives have too often failed to more than transiently modify the spasm. Fortunately, long periods of remission are not infrequent.

The muscles supplied by the facial nerve, either alone or more commonly in conjunction with other groups, may be affected by spasmodic fits, including blepharospasm, nasale spasm, grimacing, etc. These are ordinarily the external manifestation of a fixed idea, and will be described under the psychoneuroses in Part VII.

**Paralysis of the Seventh Nerve.**—Facial palsy follows lesion of the seventh nerve at any point from the convex to the stylomastoid foramen. In the five individual branches may be injured, with corresponding local motor loss. The most complete facial paralysis depends upon injury to the nerve-trunk. When it follows exposure to cold or occurs in rheumatic conditions it is often called *Bell's palsy*. The peripheral form, due to a lesion of the nerve-trunk after its entrance into the petros through the internal meatus, will first be considered, and then the nuclear and central varieties.

**Peripheral Facial Palsy.**—**Etiology.**—The facial nerve in the petrous canal is occasionally invaded by inflammation extending from the tympanic cavity. The separating plate of bone is often of extreme thinness, and may even be lacking, especially in children. Mastoid disease and pharyngeal inflammations have led to the same result. Fractures of the cranial base very frequently involve the petros, and may cause a facial palsy at once; or this may come on from inflammatory reaction after a few days, or later from the deposition of callus. In extremely rare instances hemorrhage within the canal produces paralysis almost instantly. In some infantile cases a cerebellar hemorrhage invades the canal with the same result. Injuries to the

nerve at its foramen of exit, as by blows with sharp or blunt instruments and by the obstetrical forceps, may cause the palsy. In addition to the local disadvantage presented by the facial nerve being surrounded by a long bony passage, Neumann attributes much to the large number of lymphatics and lymphatic glands encircling it at its exit from the stylomastoid foramen. Stagnation of lymph here favors changes in the neighboring nerve-trunk. Upward extension of inflammation in the parotid or side of the neck into the canal is an occasional cause. By all odds the most common and usual cause is a neuritis due to cold, as by a draft of air on the side of the face during the night, or from an open window, or after prolonged outdoor exposure. The nerve-swelling to which this gives rise nips the nerve in its unyielding channel and paralyzes supervenes. The degeneration is found throughout the manifestation of the nerve and ascends to the geniculate ganglion. The cells of the facial nucleus present varying stages of chromatolysis.

Hirschmann,<sup>1</sup> from a series of 135 cases, found seventy-five per cent. to be of the so-called rheumatic form, nine per cent. due to ear disease, and six per cent. to injury. Most of the rheumatic cases occurred between the ages of ten and forty years. Males predominated very slightly.<sup>2</sup> Reik<sup>3</sup> believes that the so-called rheumatic variety of facial palsy is very commonly dependent upon catarrhal or other inflammation of the middle ear, and urges an examination for otitis media in every case.

Any general depression of the physical state may act as a predisposing element. In this relation rheumatism occupies a chief place. Anæmia, syphilis, tuberculosis, and alcoholism are also very favoring conditions, as is the puerperium. Syphilis may of itself cause a local lesion, but must at this location act very exceptionally. Gouty and leukæthemic neuritis may also involve the facial trunk directly. Sarbo<sup>4</sup> agrees with Neumann that there is commonly a personal predisposition to facial paralysis, and Arkwright<sup>5</sup> reports six cases in two families which indicate the same thing.

**Symptoms.**—*Motor.*—In a well-marked case, unless due to direct violence, within about twenty-four hours after the action of the inciting cause a little twitching in the affected side of the face is frequently noticed. Perhaps friends then first call attention to the distortion of the face, which in all expressional movements is drawn to the opposite side. The patient may first find that he is unable to expectorate with certainty, to whistle, or to puff out his cheeks and lips, owing to the loss of control over the labial positions. In two or three days the deformity is noticeable to all. The entire side of the face is affected, with the occipitofrontalis. Even the small muscles of the external ear on the same side are paralyzed. The unopposed muscles of the sound side draw the mouth in their direction, and the zygomatic deviate its angle. When the mouth is opened widely it presents an unsymmetrical outline. It is higher on the sound side and displaced toward this side. This may mechanically cause the protruded tongue to deviate to the sound side, but if the mouth be passively held in a median position, the lingual curve disappears

<sup>1</sup>"*Neural Centralblatt*," Nov. 15 and Dec. 1, 1894.

<sup>2</sup>Waterman, "*Journal Nervous and Mental Diseases*," Feb., 1900.

<sup>3</sup>Johns Hopkins Booz. Bull., April, 1902.

<sup>4</sup>Danz. Zeit. f. Nervenk., Mar., 1904.

<sup>5</sup>"*Lancet*," Jan. 23, 1904.



and full mobility of the tongue can be easily shown. The dorsum of the tongue is slightly depressed on the affected side, according to Schultz,<sup>1</sup> from weakness of the styloglossal and digastric. Saliva and food accumulate in the flabby cheek and have to be displaced with the finger. Mastication, therefore, is mainly done on the sound side and, due largely to this fact, lateral furring of the tongue on the paralytic side may appear. Often there is some diminution of salivary flow on the paralyzed side, owing to the involvement of the secretory fibers which traverse the facial trunk. The nasolabial fold disappears on the injured side and is accentuated on the sound side. The chin and nose may deviate



FIG. 44-46.—Facial palsy of left side. 1, Bilateral attempt to close eyelids; 2, bilateral attempt to close eyes; 3, resting.

to the sound side. The platysma on the same side is invariably involved. The eye on the paralyzed side can not be closed nor the eyeball lifted. In attempts to close the eyes the eyeball on the affected side turns upward and can not be controlled. Winking is absent, and in the aged the lower lid often droops away from the ball. The cross-wrinkles on the forehead stop abruptly at the middle line.



FIG. 47-49.—Facial palsy of left side. 4, Slight effort to protrude tongue on both sides; 5, protruded tongue in middle line; 6, mouth open, lower widely on sound side in bilateral effort.

During moments of quiet little asymmetry may be apparent, but the slightest emotional or voluntary movement produces and accentuates all the motor symptoms. In young patients, where the expressional lines and

<sup>1</sup>"*Monat. und. Wochens.*," June, 1897.

wrinkles are absent or only slightly developed, the difference between the two sides is much less marked than in those of more mature years. The greater resilience of the skin and the larger adipose layer contribute to this effect. The *continua of degeneration* is present in the muscles either in complete or modified degree. The muscle reflexes are abolished and McCarthy's supra-orbital reflex is lost. After a few weeks the *muscle waste*. The atrophic loss is not readily seen except in thin people. As the eye can not be closed, the exposed conjunctiva is often irritated and painful. At night it may become quite dry, and conjunctivitis of a severe grade may develop. From the relaxation of the lids the natural flow of tears to the lacrimal duct is interrupted and they overflow on to the cheek. In rare severe cases there is a herpetic eruption, but this is probably due to a simultaneous affection of the fifth nerve.

Disturbance of *common sensation* is slight or wanting. If in the first week the muscles of the cheek be grasped, some tenderness can be elicited, which is probably due to the irritation of the sensory twigs distributed to the degenerating facial branches. The *sense of taste* on the same side of the tongue in its anterior two-thirds, the part supplied by the chorda tym-

pani, is often disturbed or abolished. Many patients during the first few days acknowledge subjective taste sensations of a peculiar sort; but ordinarily a careful search must be made in order to elicit the actual difficulty. *Hearing* is sometimes modified in such a way that low notes are more readily heard than on the sound side, while those of high pitch, as the ticking of a watch, are not distinguished with even ordinary acuteness. Middle-



Fig. 66.—Facial palsy of left side. 7, Effect of function recovered; 8, non-effort of same current on paralytic side.

ear inflammation, if present, would be attended by some general loss of hearing and sometimes by tinnitus.

In rare instances a *double facial palsy* is encountered. This usually is significant of intracranial disease, as pontine lesions, inflammation (especially of a syphilitic sort) at the base, or tubercle degeneration, which ordinarily involves other cranial nerves at the same time. A double *otitis* may produce it, or it may be part of a multiple neuritis, such as follows diphtheria and other general intoxications. In bilateral palsy the face hangs flabby, motionless, and without expression. The unliking eyes and the drooping mouth give a vicious look that is suggestive of dementia. All of the paralytic symptoms are present on both sides, but the distortions and asymmetry so prominent in the ordinary form are wanting.

*Course*.—The tendency of the palsy to improve in peripheral facial paralysis is marked, excepting in destructive lesions of the nerve-trunk. Cases that improve get practically well in from three weeks to eighteen months, depending upon the amount of injury done the nerve and the duration of pressure within the facial canal. This is true regardless of



treatment in those cases that depend upon so-called rheumatic conditions, though improvement may without doubt be accelerated by proper management. The early loss of faradic stimulability of the muscles gradually passes away, but voluntary motion returns before the faradic current regains its control. Very frequently the paralyzed muscles will twitch or even act in vigorous spasm as regeneration in the nerve restores its irritability. Those cases which last a month of two, or more, usually leave traces for the rest of life. They may be so slight as to require careful scrutiny, or marked weakness may persist. Very exceptionally the palsy is permanent.



Fig. 48.—Same man six months later. (1) shows left contraction on the paralytic side while the finger is at rest. (2) illustrates in the lower half of the face increased by gradually closing the eyes and in the same time shows weakness about left eye. (3) contraction increased by raising brows, showing contraction of supratarsal and weakness of frontalis on left side.

As the face recovers, in every instance the paralytic side is likely to overact for all moderate voluntary bilateral movements. It would seem that the neuritis had left a certain nuclear irritability, so that a central influence, acting equally on both sides, produces a greater effect on the formerly paralytic muscles. This secondary overaction is most pronounced in the lower half of the face, and often leads the patient to suppose that the sound side is becoming paralytic. When once strongly developed it practically remains for the rest of life, though slight degrees of it may pass away. While, therefore, the paralysis usually gets well, the sequel often remains and is irremediable, but is not of great amount or serious import. Indeed, many people who have never been affected show almost as much asymmetry of facial muscular action. Some cases present several attacks of facial palsy. Bernhard<sup>1</sup> asserts that recurrence takes place in 70% of all cases and in men more commonly than in women. Second attacks are frequently associated with middle ear inflammation, syphilis, or diabetes. In the authors' experience second attacks are decidedly uncommon. Rossolimo<sup>2</sup> reports a recurring case associated with migrainous attacks.

**Diagnosis.**—The diagnosis of a facial palsy, if at all marked, is made at a glance in the early stage. Later on the secondary overaction may momentarily mislead, but its nature should be easily deciphered. In slight voluntary movements the overacting and consequently fer-

<sup>1</sup> *NeuroL. Centralblatt*, 1895.

<sup>2</sup> *NeuroL. Centralblatt*, Aug., 1901.

merely paralyzed side of the face responds in an exaggerated manner; but if the muscular effort be forced, as in vigorously screwing up the eyes and in laughing, the paresis will be manifest in the lessened action of the orbicularis palpebre, though at the same time the zygomatici on the same side may be strongly overacting and exaggerating the nasolabial furrow. In the same way a strong attempt to move the teeth will show a weakness on the affected side. The distinction between a cerebral and a peripheral palsy will be made clear in the consideration of lesions within the skull. It suffices here to say that disease of the cortex and in the brain above the nuclei, as in ordinary hemiplegia, produces no muscular atrophy, no reaction of degeneration in the muscle, and, with practical uniformity, only slightly involves the lower and eyelids, but is marked in the lower portion of the face. The tongue also is affected, as a rule. Lesions at the nucleus and in the pons almost invariably implicate other cranial nerves. Lesions at the base, as in meningitis, affect other cranial nerves usually, and always, when the face is paralyzed, produce deafness from injury of the more vulnerable auditory nerve, the parallel course of which with the facial has been pointed out.

The question as to the location and extent of the lesion in the usual peripheral palsy is an interesting one, and can be answered with some degree of precision. Referring to the diagram, page 122, it is evident that the neuritis, unless confined to a very short distance above the stylomastoid foramen, will (1) involve the chorda and (2) produce loss of taste on the same side of the tongue, with diminished salivation. A little higher (3) the tympanic tensor nerve is cut off and the auditory symptoms of hyperacusis for low tones, with blunting of hearing for notes of high pitch, will be added. At the internal (4) meatus the auditory nerve will also suffer and the chorda may escape.

**Prognosis.**—The prognosis, while very good for practical recovery in all cases of facial neuritis, should always be guarded and guided by an electrical examination, which, after the second week, often furnishes information of a precise nature. Simple slight cases present no diminution in faradic response or change in galvanic excitability. They may be expected to recover within a month. If galvanism and faradism of the nerve give diminished responses and galvanism of the muscles shows increased influence, with A. C. C. equal to or greater than C. C. C., the case will probably last two or three months. Finally, if the reaction of degeneration be complete, no responses in the nerve to either current, the muscles refusing to act to faradism and showing overexcitability to galvanism with the anode overpassing the cathode, a year to eighteen months must be allowed. In this last group of cases marked and persistent overaction may and should be foretold. Boodier and Fraenkel<sup>1</sup> insist that the outward and upward deviation of the eye when the patient tries to close the lids is proportionate to the severity of the nerve injury, and recovers in an equal ratio.

Tumours of the trunk of the nerve at its bony exit, even severing it, are not necessarily of grave outlook, as union and regeneration may follow. Middle-ear diseases and cranial fractures are likely to heal and the nasolabial palsy to pass away. Naturally, the prognosis must be more

<sup>1</sup> "San. Med.," Sept., 1897.



guided in these conditions, and is based largely upon the possibility of their recovery.

**Treatment.**—The tendency to recovery being pronounced, can we shorten the duration of the disability? It seems probable. If the case is seen early, a blister or leech on the mastoid may reduce the congestion in the facial canal. Hot applications to this region may be tried. If not thoroughly and persistently used, they are worse than useless and later on quite valueless. When the case is developed we recognize that the lesion of the trunk shuts off trophic and motor control. The indication is to maintain the muscles in as perfect a condition as possible until the way for nuclear and cortical influence is reopened. Further, we should strive to keep the muscles as responsive as possible to the slight fraction of motor control that remains, or, if none is present, to the weak and inefficient impulses that will first reach them. To this end there are three things to do: (1) Keep the face in place by having the patient constantly push the cheek and mouth to their proper positions, from which every smile and word cause them to be dragged by the sound muscles. In the same way have the eyelids rubbed over the eyeball frequently, and at night a compress should be worn to keep them closed. This has the additional advantage of diminishing and often of preventing conjunctivitis. A weakened muscle stretched by its sound antagonists is not only placed at a mechanical disadvantage, but actually injured in its nutrition. (2) By means of massage and hot douches accelerate and improve circulation, and thereby the nutrition of the affected muscles. (3) By electrical stimulation of the muscles, commenced from the first day, keep up their responsiveness. For this purpose a galvanic current only is needed. Faradism to contract the muscles, or the faradic brush, are needless and painful. Ordinarily the best plan is to use the needle at the motor points on the side of the face and to daily systematically cause the muscles to gently contract six to ten times by anodal closures. This pole is the least painful and the most active in the presence of degeneration.

Traumatic cases are to be treated on surgical lines. If the nerve-trunk is divided, an attempt should be made to suture it. Beauryne,<sup>1</sup> in the manner advocated by Faure and Furet, reports a fair success following the establishment of an anastomosis between a diseased facial and the spinal accessory. This method is only applicable to those cases in which the nerve has no other possibility of regeneration. Anastomosis with the hypoglossal promises even better results.<sup>2</sup> Tympanic, nasolabial, and pharyngeal trouble will require topical measures. When the neuritis is a part of general multiple neuritis, attention is directed to the toxic cause. Guided by the rheumatic idea, such remedies as the salicylates have been recommended and may be tried if the observer can convince himself that such a blood-state exists. Anemia and diminished physical forces from any general cause are not to be neglected. Strychnin may be of some service, acting as a nuclear stimulant and general tonic.

**Nuclear palsy of the seventh nerve** manifests itself by the

<sup>1</sup> "Traumat. de Neurologie Chirurgicale," Jan., 1901.

<sup>2</sup> Ballance and Stewart, "Br. Med. Jour.," May 2, 1903; Taylor and Clark,

"Med. Rec.," Feb. 27, 1904.

same peripheral motor distribution as that found in diseases of the trunk, but lacks the gustatory, salivary, and sensory disturbances. The muscles degenerate in the same way and show the same electrical conditions. Almost invariably other adjacent cranial nuclei are implicated. This gives rise to a symptom group that easily differentiates the nuclear palsy from the peripheral form. In bulbar palsy the facial nucleus is generally invaded, and it may be affected in poliomyelitis superior. The nuclear lesion usually produces bilateral symptoms. The pyramidal tracts are also commonly affected, and symptoms in the trunk, and especially in the extremities, are present.

An *afterstroke* palsy of the face on one side and of the limbs on the other is due to a low pontine lesion (see Fig. 22) affecting the facial fibers after their crossing and the pyramidal tract above its medullary decussation.

**Supranuclear facial palsy** results from any lesion destroying the cortical facial centers or interrupting the communication of these centers with the facial nucleus. Ordinary capsular hemorrhage is, therefore, a frequent cause of facial palsy, but almost invariably at the same time causes hemiplegia. In these supranuclear palsies, as has already been indicated, the upper portion of the face partially or entirely escapes. Particularly is this true of the orbicularis palpebrarum, which acts voluntarily. The slight weakness that is present is often shown by a partial ptosis completely at variance with the constantly open eye of the peripheral palsy, but the eye on the affected side usually can not be closed as vigorously as the other. All expressional bilateral movements in the lower face may be fairly retained, while voluntary control is practically lost. In the peripheral form the loss is equal in both varieties of action.

In supranuclear palsy the muscles respond actively and normally to electricity, and their trophic condition is not impaired. In other words, the lower neuron is not involved. The reflexes are present for the same reason, and there are no auditory, secretory, or gustatory symptoms. A lesion that involves the optic thalamus may, according to Bechterew, cause loss of emotional expression on the opposite side. A lateral loss of expressional movements, with the retention of voluntary motion, therefore, points to the optic thalamus, which contains the centers for emotional expression. This loss has been found associated with corresponding bilateral hemianopsia resulting from the same lesion.

## CHAPTER VII.

### DISEASES OF THE EIGHTH CRANIAL NERVE.

**Anatomical Considerations.**—The eighth cranial nerve is purely sensory. It is made up of two portions: the cochlear branch, which alone conducts sound impressions, and the vestibular branch, which conducts space sensations from the semicircular canals. An accessory and, so to speak, adventitious portion is the intermediate nerve of Wrisberg, which is vasomotor and secretory in function. It passes to the facial nerve and finally controls salivation. In consistence the auditory stem



is much less firm than the facial. It follows that lesional lesions which affect the seventh almost necessarily involve the softer auditory, which lies beside it. But, on the other hand, a lesion that injures the auditory may not affect the more resisting facial or portio dura.

At the apparent origin of the auditory trunk the internal vestibular portion, the one related to equilibration, passes backward and inward between the restiform body of the medulla and the ascending root of the trifacial nerve, to terminate in two groups of large cells. The outer cochlear portion, the true auditory root, separates from the vestibular root near the apparent origin, and, passing outward around the restiform body, turns inward on the floor of the fourth ventricle as the stria acustica, which dip down in the middle line to the nuclear cells of Clarke. At the point where these branches diverge the cochlear root presents a group of cells, for the vasomotor root of Wrisberg, analogous to a posterior root ganglion. Another group of cells, the acoustic tubercle, often of large size in animals, is placed just outside the restiform body, and is considered a pure auditory nucleus. The higher relations of the auditory nuclei are not clearly known, nor the exact course of the radiations to the cortex. There is every reason to believe that the vestibular nuclei are in relation with the cerebellum and with the cerebrum. The cochlear nuclei are connected with the temporo-sphenoidal cortex by fibers which pass through the posterior third of the sensory division of the internal capsule.

Each ear is represented on both sides of the brain, but also principally upon the opposite side. The auditory center for speech, however, in right-handed individuals is practically confined to the left temporal lobe, the destruction of which produces word-deafness, or a loss of spoken-word memories. At its peripheral termination the auditory nerve enters the cribiform opening in the internal meninges. The auditory portion is distributed to the cochlea and the organ of Corti. The labyrinthine portion is distributed to the vestibule and ampullæ of the semicircular canals.

**Irritation of the auditory portion of the eighth nerve** is marked by *auditory hyperæsthesia*, by increased acuteness of hearing (*hyperacousis*), and by *trinnitus*. It must be understood that continued irritation of the auditory apparatus may result in diminished or lost function, just as overstimulation of any tissue or organ finally produces weakness and involution. It follows that tinnitus, for example, is often found with defective hearing. *Hyperacousis* is frequently present in excitable and nervous persons. Migraine and all forms of pain often render the sufferer more sensitive to sudden noises, especially of high pitch, but

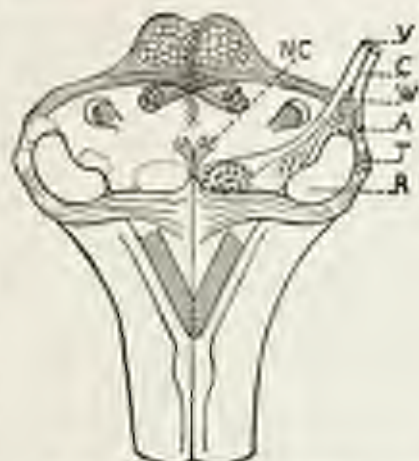


Fig. 30.—Diagram of a section showing the origin of the eighth cranial nerve: V, Vestibular portion; C, cochlear portion; W, nucleus of Wrisberg; A, acoustic tubercle; T, stria acustica; R, restiform body; NC, nucleus of Clarke (after Edmond).

actual hyperacusis is rare. In meningitis, acute mania, and under the influence of some stimulant drugs, such as alcohol, opium, anesthetics, Indian hemp, and coffee, the special sense of hearing is at times actually exalted. Stoddart insists that the hearing is often abnormally acute in maniacal patients. It is also conceivable that an irritant lesion of the auditory cortical centers might give rise to hyperacusis, and perhaps this is also the explanation of some hallucinations of hearing and sometimes of the rare auditory aura of epilepsy. The increased acuteness of hearing for low tones in facial palsy has already been described.

Irritation or disease of any part of the auditory mechanism is likely to produce a tinnitus which may vary widely with the cause and with different cases. It may resemble hissing, roaring, buzzing, singing, ticking, throbbing, the sound of bells, and every conceivable monotonous noise. Cerumen or foreign bodies in the external meatus, inflammation of the middle ear, Eustachian occlusion, the throbbing of the carotid in its canal in the petros, rhythmical clonic spasm of the palate and orifice of the Eustachian tube in hysterics, neurological irritation of the auditory nerve-trunk, perforations of the drum-head, sclerosis of the internal ear, and many other conditions may be symptomatized by a tinnitus aurium. A tinnitus is present in some cases where aurists are unable to detect any peripheral cause, and persists practically unchanged throughout life. A slight tinnitus aurium is so commonly found as almost to be invariably present after the fifth decade of life. As a rule, a continuous tinnitus interferes with hearing, which is thereby diminished. Fortunately, it is frequently unilateral. Certain drugs, as quinin and the salicylates, cause tinnitus, and quinin in large and repeated doses has caused permanent deafness. A careful aural examination is needed in every case of tinnitus, and the reader is referred to the special works on ear diseases. The treatment is that of the inciting condition.

**Paralysis of the auditory nerve** and unilateral deafness may follow a cortical lesion involving the temporal lobe. Memories of heard speech seem to be stored up in the first left temporal convolution. A destructive lesion here is followed by word-deafness, and the patient no longer understands what is said, though he clearly hears the tones of the voice and all sounds. A bilateral lesion of the temporal cortex causes complete deafness. Lesions in the sensory portion of the capsule may cause a unilateral deafness on the same side with the accompanying hemianesthesia. A unilateral loss or diminution of hearing in the hemianesthesia of hysteria is not uncommon. It may come on suddenly and disappear in the same manner. The presumption is that the cortex is functionally at fault. Complete—that is, bilateral—hysterical deafness is seldom encountered. Nuclear disease of the eighth nerve is practically unknown, though tumors of the restiform body<sup>1</sup> and the medulla may involve the cochlear root.

According to Virchow, the auditory trunk, most frequently of all the cranial roots, is the seat of new growths. Tumors at this point give rise to the general symptoms of brain-tumors, but are capable of definite diagnosis in many instances. According to Fraetkel and Hunt,<sup>2</sup> who have collected nine cases, the symptoms may be arranged

<sup>1</sup> Bristoll, "Leçons sur les Maladies Nerveuses," Paris, 1895.

<sup>2</sup> "Ann. of Surg.," Sept., 1904.



as follows: (1) General: Headache, vertigo, vomiting, optic neuritis, leadcardia. (2) Focal: Peduncular ataxia, cerebellar ataxia, lateropulsion, hemi-synergy, homio, contralateral and crossed paralyses of the extremities, paralysis of the basilar cranial nerves, especially the seventh, sixth, and fifth, dysarthria, dysphagia, nystagmus, paralysis of the conjugate movements of the eyes, inequality of the pupils, and attacks of Adam-Stokes syndrome. (3) Special: Serious impairment of the function of the auditory nerve, of long standing and gradual onset. 'Cushing' in a monographic review additionally emphasizes suboccipital pain, tilting of the head to the affected side, nystagmus increased on looking toward the affected side, and loss of the corneal reflex on the affected side.

Gouty deposits and hemorrhage in the substance of the root have been found. It very frequently is involved in syphilitic meningitis. In purulent meningitis infection may travel along its sheath into the inner ear and produce deafness that is usually permanent. After exposure to cold the eighth nerve is sometimes subject to a neuritis similar to that so common in the facial, which may or may not be associated in the process. The condition is marked by diminished or complete loss of hearing, but is of favorable prognosis. Artisans who work constantly amid loud noises—as boiler-makers, tin-smiths, and other metal workers, engine-drivers and firemen on railroads—often present a partial loss of hearing that may progressively increase. They sometimes hear better in the accustomed turmoil than in quiet places, and this is also true, but much less commonly, of ordinary deafness. The auditory nerve may be injured within the petrous by the extension of inflammation from the mastoid, tympanum, or pharynx, and by basilar fractures.

**Diagnosis.**—The first thing to be settled in a case of deafness is the integrity of the nerve. If the nerve is at fault, the condition is called *nervous* or *nerve deafness*. When there are no basilar symptoms, involvement of other cranial nerves, bulbar or cerebral indications, and when sounds of high and low pitch are not heard through the air or by bone-conduction, we may safely locate the disease in the nerve. Rinne's test (see p. 65) enables us, when the hearing is reduced, to fairly determine whether the difficulty is in the conducting apparatus or in the nerve. A great reduction of hearing, in which air-conduction remains better than bone-conduction, but in which both are deficient, points to nervous deafness. Lesions within the brain-stem and in the temporal lobe must be determined by the association of symptoms peculiar to those localities.

**Treatment.**—In the treatment of nervous deafness we have first to investigate the aural apparatus and remove, if possible, any diseased conditions that may be present. All acute inflammatory troubles must subside before active measures are instituted. The use, then, of strychnin, preferably hypodermatically, in much the same way as for optic atrophy, can be recommended. Electricity has small claims to notice, though usually suggested. Unfortunately, very little improvement can be expected. Nerve deafness of sudden onset, whether due to syphilis, neuritis, congestion, or hemorrhage into the internal ear, is sometimes favorably modified by the use of pilocarpin in full doses twice daily for a week or two.<sup>1</sup> Free action on the skin is to be produced. In these

<sup>1</sup> *Tumors of the Nervous System*, W. H. Saunders Co.

<sup>2</sup> *Danforth's Grant, "Brit. Med. Jour.," Nov. 16, 1896.*

cases quinin and the salicylates are contraindicated, though sometimes of value in the chronic forms.

**Disease of the Vestibular Portion of the Eighth Nerve.**—*Aural Vertigo, Ménière's Disease.*—The function of the semicircular canals is to furnish information relative to position in space. This information, of course, is correlated with similar information derived through sight, touch, joint sensation, muscular sense, and perhaps even to a slight extent through hearing. The canals are the principal organs of spatial orientation. Placed in three different planes they act much like three small spirit levels. Any movement, either active or passive, of the endolymph and the otoliths against the sentient nerve-endings produces a stimulus which is transmitted to the brain by the vestibular branch of the eighth cranial nerve. The flow of endolymph may be occasioned by movements of the head, modifications of pressure within the labyrinth, or decided changes of temperature causing thermal currents in this fluid. Certain toxic and autotoxic substances may also cause vertigo by upsetting these space organs or their nervous mechanism. It is only necessary to mention tobacco and alcohol.

It has long been recognized that disturbance in the internal or middle ear, and even in the external meatus, may cause not only tinnitus, but vertigo. The sudden inflation of the drum through the Eustachian passage, the use of interrupted galvanic currents about the ear, and any instrumentation within the tympanum or the external meatus may produce giddiness. If destruction of the vestibular branch takes place, the vertigo ceases. In cases of aural vertigo external objects may seem to revolve to the right or to the left. In other instances the vertigo is subjective, and the patient feels as if revolved to the right or left or as if falling forward, backward, or downward. The intensity of the vertigo varies greatly. It may be quite insignificant or it may be so pronounced that the patient holds to any neighboring person or object, staggers, or is even forcibly projected in some given direction. The attacks are usually paroxysmal, with relative freedom in the intervals. In the form described by Ménière the victim is struck down as if shot, there may be unconsciousness for a few moments, and the patient is often pale and covered with perspiration. Nausea and vomiting may occur.

Attending aural vertigo there is an associated nystagmus which corresponds to the ear and even to the semicircular canal that is affected. This nystagmus consists of lateral, vertical, oblique or rotary rhythmical movements of both eyeballs made up of two phases: (a) a slow movement in one direction and (b) a quicker return. Voluntary deviation of the eyes in the direction of the quicker movement accentuates the nystagmus, and, *per contra*, deviation of the line of vision in the direction of the slow movement diminishes the nystagmus. The variety of nystagmus is named according to the quick movement. Thus a right nystagmus is one that presents the quicker movement to the right.

When aural vertigo is attended by its associated nystagmus the person so affected tends to move and even to fall in the direction of the slower nystagmic movement. At such times if he try with closed eyes to touch a given object with his finger he points to one side of it, and always to the side corresponding to the direction of the slow movement.



The great majority of cases of aural vertigo occur after thirty years of age. In childhood they are extremely infrequent and rapidly increase after middle life, men being affected twice as frequently as women. Gout, rheumatism, and the sclerotic changes of old age are frequently causal. These may act directly upon the labyrinth or indirectly through the blood-supply and the pressure of the endolymph. A vasomotor element is given considerable importance by some. The irritation of the vestibular filaments is usually associated with auditory phenomena, so that tinnitus and defective hearing are almost invariably present. The close anatomical relations of the two portions of the eighth nerve explain this. In some cases the labyrinth has been found the seat of hemorrhage or local disease. Sometimes its epithelial structure is degenerated. Usually disease of the middle and external ear is wanting in the severest cases, while decreased hearing and tinnitus point strongly to involvement of the nerve itself. Some of the cases of the Ménière type show a progressive tendency, with failing hearing, first in one, then in the other, ear. Complete physical disability may ensue, due to the vertigo and attacks of falling. In some instances the disease remains at a standstill for years, and may even recede and hearing be restored. Again, when hearing is lost the vertiginous attacks may cease. Milder varieties run various courses, depending upon their causation and other conditions.

Diagnosis of an aural vertigo depends in practice partly upon the association of auditory symptoms. Tinnitus or defective hearing, or both, are ordinarily present. The defect in hearing, as tested by bone-conduction, is sometimes unexpectedly great, the ticking of a watch when placed on the mastoid being inaudible. If the vertigo is produced or increased by changing the air-pressure in the tympanum, as by firmly pressing the tragus into the mentus or by Politzerization, the significance of that fact is great. Frequently the attack of vertigo is associated with an intensification of the tinnitus, or there are subjective sounds of a violent character, described as "pistol shots," "something breaking in the head," etc. Sometimes a quick movement of the head, as in turning over in bed or in a given direction, produces it. This apparently has relation to a particular semicircular canal, which is mainly or alone affected. A further characteristic of aural vertigo is the fact that the subjective or objective gyrations are uniform in the given case, or the stagger or falling is always in the same direction. In epilepsy we not infrequently encounter an indescribable vertiginous aura, but never the formulated vertigo of aural disease. The epileptic attack is usually followed by mental hebetude, which is lacking in aural attacks, where the vertigo may be maintained for a long time, giving rise to distressed feelings, vomiting, and collapse. The sensorium is always clear in aural vertigo, excepting the initial momentary unconsciousness of the severest form, or in the delirium that a continuation of the extremest variety may produce in very rare instances. The vomiting usually gives rise to the idea of "bilioousness," and frequently a brisk cathartic, relieving all the symptoms of vertigo, is supposed to confirm the idea of intestinal or hepatic derangement, its influence on cerebral circulation being over-

locked. Again, the sudden onset of the attacks in the *Ménière* form suggests cerebral disease or earlike attacks, to which mistake the age of the patient and his arterial degeneracy often conduce. The repetition of the nural attack during periods of rest, and even during sleep, with absence of earlike and cerebral symptoms in the meanwhile, should correct a misconception of this character.

In recent years a number of valuable labyrinth tests have been developed based upon the association of vertigo and nystagmus. Exahl found that he could produce in animals a definite rotation of the head with a definite nystagmus by making pressure on a given semicircular canal, and opposite movements by reduction of endolymph pressure. In man when there is a fistulous opening into the internal ear, similar pressure conditions induced by hand-bullb apparatus may cause identical results if the vestibular nerve is intact. Following the turning methods of Bárány, if a normal person with closed or covered eyes, the head erect, be turned to the right about his vertical axis ten times in twenty seconds, upon stopping the rotation a nystagmus will be observed in both eyes. During the rotation the nystagmus is to the right, but upon arresting the rotation it becomes a left nystagmus. Mechanically, in turning a patient to the right, as in a revolving chair, the endolymph in the horizontal canals lags behind and this gives a definite stimulus associated with spacial experiences aroused by such movements and a right nystagmus. Upon suddenly arresting the rotary movement the endolymph surges to the opposite end of the canal and an opposite nystagmus is produced, which lasts twenty to forty seconds. As it is difficult to observe the nystagmus during rotation, the secondary nystagmus is the one commonly noted in these tests, and is the one always understood unless otherwise specified. Apparently the nystagmic movements are ocular attempts to re-establish spacial orientation.

In these turning tests vertigo is also experienced. When the rotation is stopped the patient still feels as if being turned to the right, and with closed eyes points to the right of any object and tends to fall to the right, that is, in the direction of the slow nystagmic movement. Reversal of the rotation reverses the nystagmus in every particular, unless there be one-sided labyrinthine disease, when the corresponding nystagmus will be proportionately reduced. With the head placed in various positions, so as to bring the other canals into the plane of rotation, similar vertigos and nystagmic movements are induced. If, however, the semicircular canals or the vestibular nerve be diseased the nystagmus is much reduced, and upon the destruction of the canals or division of the vestibular nerve it does not occur at all.

As both ears and both eyes are in mutual and bilateral relation in the function of physical orientation, these turning tests do not enable one to test each ear separately, but are highly valuable in conditions such as otosclerosis affecting the internal mechanism or the nerve supply of both ears.

Thermic tests have the advantage that they can be applied to each ear alone, and by changing the position of the head any canal may be examined singly. Water 20° F. above or below the body temperature is slowly injected into the meatus, freed of any local obstruc-



tion. The warm water sets up currents in the endolymph which, of course, are reversed by cold water. The effect of the cold water (78° F.) in one ear produces nystagmus to the *opposite* side, while warm water (118° F.) causes nystagmus to the *same* side. Vertigo follows the nystagmus in a few moments. The cold water produces its nystagmic effect in about thirty seconds, and the ocular movements persist much longer than after the turning test, owing to the prolonged thermic effect on the bones and soft parts. Such effect must be allowed to pass before the test is repeated on the same or opposite side, or it can be terminated by an aural douche of water at body temperature. In order to get the best results from the thermic test the patient, if sitting up, should bend the head well backward at an angle of about 60 degrees. The test is measured by the promptness with which the nystagmic reaction is produced and its duration. Any lesion of the vestibular tract, from nerve centers to end organs in the semicircular canal, causing reduction of functional activity, may give a corresponding reduction of activity to this test, and, *per contra*, any such lesion may be attended by a constant or intermittent nystagmus. According to Neumann it is possible to differentiate between the nystagmus of cerebellar disease and that of the labyrinth, in that the cold-water test causes a nystagmus to the *same* side in the cerebellar lesion. For more elaborate descriptions of labyrinthine tests and diseases the reader is referred to special works on ear disease.

The labyrinthine variety may be readily confused with ocular vertigo in some instances, as it occasionally gives rise to persistent nystagmus, and even has produced diplopia. The patients sometimes describe oscillating movements in viewed objects, rapid in one direction with slow return, similar to the nystagmic movements of the eyeballs. The mutual dependence of space sensations and ocular impressions only needs to be mentioned to explain the secondary ocular movements. Ocular vertigo ceases the moment the eyes are closed, but this has no effect on the aural form.

Vertigo is associated with numerous abdominal disturbances, particularly those of the stomach, liver, and small intestine. These forms of vertigo are usually attended by indigestion or other symptoms of a local character, and the vertigo commonly lacks the distinctive gyzatory feature of ear trouble. In some cases of aural vertigo, however, the patients complain merely of "dizziness," "giddiness," or "swimming" sensations. If the vestibule alone is involved, without any implication of the cochlea, as is conceivable, all audition symptoms default. In such cases the diagnosis must largely depend on the exclusion of other sources of vertigo.

**Treatment.**—If aural vertigo is recognized as an irritation symptom its rational treatment will depend on appreciating and, if possible, removing the basic disease. As practically every vertigo is labyrinthine, even when caused by intestinal, cardiac, vascular, and toxic causes, all such primary factors must be removed or controlled. Some cases may be relieved or even cured by Politzer's inflation, by the removal of cerumen, or by the correction of a pharyngeal catarrh. In others the sclerotic changes in the labyrinth are irremediable and treatment is directed to

reducing the irritability by bromids. Charcot strongly recommended in the Ménière type the use of quinin in large doses, but others have not had his success with that drug, and it should not be used in nerve cases. He even advocated the destruction of the inner ear, producing loss of hearing, or, in other words, a removal of all irritability and the cessation of the vertigo at once, in the same way that sometimes occurs naturally. This has been practically applied by Milligan<sup>1</sup> with favorable results in three cases. Removal of the malleus and incus and mobilization of the stapes have given relief in many instances and may be advised with propriety, especially if the hearing is greatly impaired. Intracranial division of the auditory nerve has been done (Krause, Frazier), and in suitable cases of great severity should be recommended. When the vestibular apparatus is destroyed by surgery or disease the other avenues of spatial perceptions seem to answer every purpose. Gout and arteriosclerosis, middle-ear disease, and lesions of the auditory stem must be treated in their own several ways. Electricity is of questionable value, though it is asserted by some that the positive pole over the tragus and the negative on the back of the neck, with a current of three or four milliamperes gradually increased from zero, continued for five minutes and then decreased, has a quieting influence. All interruptions should be avoided. Cases of acute onset are sometimes benefited by pilocarpin, as in nervous deafness, with which they are usually combined.

## CHAPTER VIII.

### DISEASES OF THE GLOSSOPHARYNGEAL, VAGUS, AND ACCESSORY NERVES.

**Anatomical Considerations.**—The glossopharyngeal and pneumogastric nerves and the bulbar portion of the spinal accessory should be considered as one mechanism. Their nuclei in the medulla are practically inseparable, and they continuously furnish sensation and motion to the gastro-intestinal tract from the pharynx to the duodenum. In addition they supply motor filaments, all of which come from the accessory portion, to the lungs, larynx, and heart. They are visceral nerves. It is to be kept clearly in mind that the spinal portion of the accessory is a pure motor nerve to the skeletal muscles of the neck, and is only locally associated with the pneumogastric. The interrelations of the glossopharyngeal-vagus-accessory group are so complex, their distribution so wide-spread, and their indirect disturbances so vague that they furnish many perplexities. A short outline of the glossopharyngeal is first given, and then the vagus and true accessory are discussed together.

#### DISEASES OF THE GLOSSOPHARYNGEAL NERVE.

The ninth cranial nerve is still a source of anatomical contention and physiological doubt. In consequence its diseased conditions are uncertain and obscure. Practically, in man, it is never alone diseased. If its relations to other cranial nerves are considered, this fact is readily

<sup>1</sup> *Brit. Med. Jour.*, Nov. 5, 1904.



understood. Through Jacobson's nerve it forms, with the sympathetic, the tympanic plexus, whence a branch connects it through the Vidian with the facial nerve, and another branch through the small superficial petrosal connects it with the otic ganglion. It is connected with the pneumogastric at the petrosal ganglion of that nerve, and also in the pharyngeal plexus. Its nuclei are intimately associated with those of the vagus and accessory nerves. It probably subserves sensation in the upper part of the pharynx and in the tympanum, and nausea is associated with its disturbance. Probably through its distribution to the root of the tongue it peripherally carries the fibers of the special sense of taste for that area, but these are not embraced in its root. They reach the brain by a circuitous route, probably entering the petrosal ganglion of the glossopharyngeal nerve from the middle branch of the fifth through the tympanic plexus and otic ganglion. It seems to have some motor control of the upper portion of the pharynx, and, perhaps, of the palate.

Intracranial disease and cranial fractures may implicate the glossopharyngeal, causing weakness and some insensitiveness in the upper pharynx and in the palate. Its nuclei in the medulla usually suffer in bulbar palsy, and thus are produced, at least in part, the pharyngeal symptoms of that disease.

#### DISEASES OF THE VAGUS AND BULBAR PORTIONS OF THE ACCESSORY.

**Pharyngeal Branches.**—The pharyngeal branches of the pneumogastric follow below the glossopharyngeal, and with it form the pharyngeal plexus, supplying motion and sensation to the uppermost portion of the intestinal tube. These branches are paralyzed by nuclear disease and in diphreric palsy, but seldom otherwise. Bulbar involvement is invariably attended by symptoms in other cranial nerves. The pharynx is then more or less insensitive and motionless. The pharyngeal reflex is lost. Food tends to accumulate and lodge in the gullet or overflows into the larynx, producing spasmodic cough and strangling. If the palate at the same time is weakened, food and fluids may be forced into the nasal passages and regurgitate through the nose. A pharyngeal spasm furnishes the condition commonly noted in hysteria as "globus," or *oesophagismus*, and is practically always functional. At times it may be mistaken for pharyngeal paralysis, or the difficulties in swallowing in the latter may be attributed to spasm. The use of a sound will at once clear the doubt. The decided pharyngeal grasp of health is increased in spasm and lost in paralysis. Moreover, spasm is temporary or recurrent, and paralysis is continuous.

**Laryngeal Branches.**—The larynx is innervated by two branches of the pneumogastric: (1) The *superior laryngeal* governs the movements of the epiglottis and controls tension in the vocal cords through the cricothyroid, which is the only intrinsic laryngeal muscle supplied by this nerve. It also furnishes sensation to the larynx above the vocal cords. (2) The *recurrent laryngeal*, which turns about the aorta on the left side and the subclavian artery on the right side, supplies sensation

to the trachea and to the larynx below the vocal cords. It controls all the intrinsic laryngeal muscles except the cricothyroid. These muscles have three principal vocal actions: First, to draw the vocal cords taut; second, to bring them close together; third, to draw them apart. Though many laryngeal movements are highly complex, requiring the synergic action of several groups of muscles, it is well to remember that the *chief tensor* are the cricothyroids, the *chief adductors* are the posterior crico-arytenoids, the *chief abductors* are the lateral crico-arytenoids. In addition, the thyro-arytenoids, which in part form the vocal cords, serve to stiffen them and make their apposition uniform and effective. By some they are considered tensors and by others laxors of the cords, and probably serve both purposes.

Laryngeal paralyses vary in degree and in distribution. They may be unilateral or bilateral, partial or complete. Further, the adductors, the abductors, or the tensors of the cords may be alone or mainly involved. Abductor paralysis is, however, by far the most common,<sup>1</sup> even when the lesion falls upon the recurrent. A full knowledge of the anatomy and mechanism of the larynx is required to understand this subject, and the use of the laryngoscope is requisite for exact diagnosis. The following table is given to show the common varieties of laryngeal paralysis, with diagrams of the corresponding mirror pictures, which should be compared with the normal outlines in phonation, respiration, and death.

LARYNGEAL PARALYSES.

Form of Paralysis.	Muscles Involved.	Cause.	Effects.
Bilateral adductor paralysis. (Fig. 34.)	Both lateral crico-arytenoids and the arytenoides.	Anæmia, physical weakness, hysteria.	Voice lost, but cough and laugh possible; respiration and swallowing normal; no pain.
Unilateral adductor paralysis. (Fig. 35.)	One lateral crico-arytenoid.	Toxæmia, lead, diphtheria, cerebral disease, small-pox, syphilis, phthisis.	Diminished voice; hoarseness; coughing, laughing, and sneezing diminished; difficulty in swallowing occasional.
Bilateral abductor paralysis. (Fig. 36.)	Both posterior crico-arytenoids.	Toxæmia, hysteria rarely, injury to both recurrent nerves, as by enlarged bronchial glands; diseased thyroid, new growths in the neck, etc.	Voice little affected by ordinary efforts; respiration impeded; extreme respiratory stridor, with low expiration.
Unilateral abductor paralysis. (Figs. 37 and 38.)	One posterior crico-arytenoid.	Stretching or injury to one recurrent nerve, as by acute meningitis, and the same causes as in the bilateral form, acting on one side only.	Voice hoarse, impaired, and hoarse; some respiratory stridor.

<sup>1</sup> Simon, "Brit. Med. Jour.," Jan. 1, 1898.



## LARYNGEAL PARALYSIS.—(Continued.)

Form of Paralysis.	Motoric Disorder.	Cause.	Remarks.
Bilateral paralysis of larynx.	Corditis.	Cold; voice straining; diphtheria.	Hoarseness; inability to take high notes.
Paralysis of the cords proper. (Figs. 50 and 59.)	Thyroarytenoids.	Overexertion; hysteria.	Loss of elastic action and uncertainty of voice; usually attended by some adductor spasm, and frequently by loss of power of the arytenoids.



Fig. 51.—Normal phonation.



Fig. 52.—Normal deep inspiration.



Fig. 53.—Normal exhalation.



Fig. 54.—Bilateral adductor paralysis. Attempted phonation.



Fig. 55.—Left adductor paralysis. Attempted phonation.



Fig. 56.—Bilateral adductor paralysis. Deep inspiration.



Fig. 57.—Left adductor paralysis. Deep inspiration. Affected cord in adductor position.



Fig. 58.—Left adductor paralysis. Phonation. Affected cord in adductor position. Right cord closing median line.



Fig. 59.—Bilateral thyroarytenoid paralysis.



Fig. 60.—Bilateral thyroarytenoid paralysis and paralysis of arytenoids, giving an incomplete squaring.

In complete bilateral paralysis of the larynx, such as results from injury to both recurrent nerves, the vocal cords occupy the cadaveric position, and are motionless. There is no voice, and coughing and sneezing are impossible. Deep inspiration develops stridor. If the complete paralysis is unilateral, the motionless, paralyzed cord occupies the cadaveric position, while its fellow moves actively in phonation and respiration, even passing the middle line in adduction. The voice is low-pitched and hoarse, cough is absent, and stridor only appears on very deep inspiratory efforts.

The treatment of laryngeal palsy depends on that of the causative condition. Laryngeal palsy due to surgical injury of the nerve, as by the inclusion of the pneumogastric in ligation of the carotid, or its division in operations on the thyroid, have occurred. Here the immediate danger is to the heart. In the diphtheric forms and in other toxic varieties, the use of electricity is recommended. To be of any value it must be applied with the intralaryngeal electrode and requires special skill. The neuritic forms of laryngeal palsy are of fair prognosis, with or without treatment, if the patient survives the early effects of the toxemia. The nuclear varieties are practically irremediable.

Anæsthesia of the larynx is occasioned by interference with the superior laryngeal nerve, which supplies sensation above the vocal cords. It may be unilateral or double-sided. In hemianæsthesia from cerebral lesions and hysteria it may be unilaterally present, with preserved reflexes, which are lost in nuclear or trunkal disease of the pneumogastric. Hysterical adductor paralysis with aphonia commonly presents a loss of sensation at the laryngeal inlet.

Laryngeal spasm is due to irritation of the recurrent laryngeal nerve or to reflex causes mainly arising in the pneumogastric periphery and acting through this branch. With the reflex action that protects the larynx from the entrance of foreign bodies we are familiar. This may be intensified by local irritation, as in catarrhal laryngitis, especially in children, giving rise to croupy cough and attacks of croup at night. The laryngismus stridulus of rickets, or tetany, or enlarged thyroids, or in goitrous, pubescent girls, is due to adductor spasm. It may be dependent upon a long uvula, enlargement of the pharyngeal tonsil, or nasal conditions sufficient to provoke the reflex. Indigestion, especially in children, and more particularly in ill-nourished children, is a common source of reflex laryngeal spasm. It may, in adults, be the result of injury to the nerve. It not uncommonly is the acoustic equivalent of asthma or migraine, with which it may alternate. The epileptic cry is due to a laryngeal and thoracic spasm. There is a variety that is sometimes called laryngeal epilepsy. It may be an element in hysterical convulsions, or the only representative of such attacks.

A partial laryngeal spasm in severe stammering sometimes induces an explosive utterance attended by evident respiratory difficulty. From overuse of the voice, especially by bad methods, a spasmodic neurosis of the larynx similar to a writer's cramp may be set up. Speech at first is fairly uttered, but the unbalanced and spasmodic action of the vocal apparatus promptly manifests itself by loss of modulation and by ex-



plosive emuciation. Laryngeal spasm is marked by a sudden onset and often comes on during sleep. There is every evidence of dyspnea, and the marked stridor, both on inspiration and expiration, serves to distinguish it from abductor palsy of the larynx. The attack is very short, lasting but a few seconds at most.

A number of spasmodic nervous coughs are described, such as the barking, explosive, incessant cough of hysteria, the metallic ovarian cough of young girls, and the barking cough of pubescent and maturing boys. In all of these conditions there is a strong nervous element that must be given first importance in etiology and treatment.

**Pulmonary Branches.**—The pulmonary branches of the pneumogastric with branches from the sympathetic ganglia accompany the bronchi into the lungs. It is probable that the vagus supplies motor filaments to the bronchial muscles of unstriped fiber. The nutrition of the lungs also seems to be under their control, though the vasomotor supply comes through the sympathetic. It has been noted in animals that death, after division of the vagus, is due to pneumonia. The principal pneumogastric pulmonary derangements are bronchial asthma and protracted hiccup, which in some rare cases are interchanged.

**Bronchial or Spasmodic Asthma.**—The early contention of Tissot, Williams, and others, that there occurs a spasm of the bronchioles in asthma, is confirmed by Bert and proven by Berner. It is accompanied by turgescence of the mucosa and a characteristic exudate of mucus in the form of spirals, which often contain polygonal crystals, by Leyden supposed to be causal of the attack. There can be no doubt that this neurosis is common in some families by direct inheritance for generations. In rare cases in the same patient it has alternated with migraine or attacks of hiccup, and even with epilepsy. In other instances, again, in neurotic stock it has taken the place of graver neuroses and of the psychoses in members of the same or succeeding generations. While it may originate apparently without any cause extraneous to the individual, in many cases every attack can be traced to certain irritant factors, such as the inhalation of dust, the pollen of certain plants, or a certain odor. It has been shown by Hack and confirmed by many others that nasal conditions may incite and prolong the attacks, which do not appear when the source of nasal reflex irritation is removed. In the same way intestinal and genital disturbances may, in individual cases, be the starting-point of the attacks. Spasmodic asthma is also associated with defective renal activity and the various acute and chronic uræmias. Its relation to gout, rheumatism, and plumbism is not less clear. In every instance a potential state must be present, and these peripheral or endogenous excitants merely fire the train resulting in the nervous explosion of the asthmatic attack.

Spasmodic asthma, except in the form of hay asthma, rarely appears before adult age. It is more frequent in men than in women, a fact that is to be explained by their greater exposure to the inclemencies of the weather. There is no doubt that laryngeal and bronchial irritation from such cause may be provocative of the asthmatic attack. In

long-standing cases the secondary pulmonary emphysema and chronic bronchitis constantly keep the spasmodic features within striking distance.

**Symptoms.**—Asthmatic attacks come on, as a rule, with considerable suddenness, and are marked by intervals of practically complete relief. Except in the hay-asthma varieties, the patient most frequently is awakened from sleep by distressed breathing that rapidly grows worse. Inspiration and expiration are both difficult, and expiration is greatly lengthened. As the dyspnea increases and persists the face is suffused and the lips become bluish. The patient is covered with perspiration and evidences his distress only too plainly by the laboring chest, the anxious and drawn face, and the noisy, blowing, wheezy respiration. The thorax is rounded, the diaphragm depressed, and its excursions diminished; the muscles at the neck are in strong relief in the attempt to increase the respiratory action. The patient calls for air. He often props himself up in bed or on chairs and other objects to raise and fix the shoulders, thereby increasing the action of the intercostal respiratory muscles. At last, when he seems unable to endure longer, the spasmodic breathing lessens, relief is experienced, and he may fall into the sound sleep of exhaustion, perhaps to be again awakened after a few hours by a repetition of the attack. The first attacks are usually mild, and only attain the indicated intensity after a number of asthmatic bouts. In the advanced and ancient cases a small degree of spasm may be continually present, manifesting itself upon the slightest exertion or exposure. During the attacks the physical signs are very slight. Roughened bronchial breathing and moist rales are noted. At first hard coughing brings up a little mucus, but toward the end of the attack a considerable quantity is frequently ejected with apparent ease and relief.

**Treatment.**—In the management of asthma the neurotic condition should ever be kept in mind. General measures to build up the tone and stability of the nervous system are essential. Outdoor life and moderate exercises, hydrotherapy, tonics, correct habits, and hygienic measures are indicated. The exciting element must be most carefully and persistently sought. If this can be recognized early, its removal gives hope of complete immunity from the recurring attacks that in so many cases make life wretched. It is probable that every repetition of the asthmatic storm reduces the power of resistance and serves to establish an increasing asthmatic habit. When this has been formed, and bronchitis with emphysema induced, a cure can not reasonably be expected. Regarding drugs, the iodid of potassium has a deserved reputation. It is particularly indicated in the chronic and uremic forms of asthma. Strychnin in large and increasing doses gives occasional aid. It may be given in doses increasing to  $\frac{1}{16}$  of a grain three times a day, if well tolerated. The correction of peripheral states in the nose, intestines, kidneys, genitals, etc., has been sufficiently urged. Climate changes are of importance, but one can never predict the result of this measure. In every case it is experimental. One patient will have complete immunity in a locality that is unbearable to another apparently similar case. In hay asthma a patient may secure relief one year at some resort and the following season find his journey futile.



The attack can often be cut short by the use of any one of a number of sedatives. Inhalations of nitrite of amyl or chloroform may give almost instant relief. Fumigations with niter and stramonium or some similar solimaceous plant are much in favor. Cigarettes and pestiloes of such materials are prepared by the trade. The smoke must be deeply and freely inhaled. A dose of spirit of chloroform or of sulphuric ether is often productive of temporary benefit. Inhalation of steam charged with camphor is a ready and sometimes efficient measure. This is furnished by dropping a dram of any camphor preparation into a pint of boiling water in a small pitcher, over which the patient holds his face.

Hiccup is usually referred to the phrenic nerve, the diaphragmatic motion being considered its most important feature. It appears, however, to be a respiratory difficulty, and is undoubtedly associated with the respiratory centers. Not only does the diaphragm act spasmodically, but there is a general thoracic inspiratory movement and a laryngeal fixation or spasm, giving rise to the peculiar inspiratory sound with which all are familiar. In some cases there are protracted attacks of hiccup that are accurate equivalents for asthma or other nervous disturbances. Such attacks may also occur independently. Injury to the phrenic or to the pneumogastric, or even to the superior laryngeal nerve, has occasioned it. In hysteria it is not a rare manifestation, and may last for weeks during the waking hours. It may be dependent upon distant reflex irritation in the intestine or genito-urinary tract, or upon affections of the larynx and pharynx. In all conditions when the respiratory centers are intoxicated or depressed, as in uremia, syncope, suffocation, after hemorrhage, in cholera, etc., hiccup may appear, and is of serious significance.

The treatment of a protracted attack of hiccup is etiological. Some rebellious cases have been promptly terminated by inducing vomiting, which is the exact opposite of dysphagia. Sedatives of all sorts have at times seemed useful, and musk is especially recommended. Faradism to the phrenic nerve and diaphragm has served a good purpose. In the hysterical cases often nothing short of complete isolation and the most thorough management of the hysterical condition will avail.

**Cardiac Branches.**—The cardiac plexus is made up of accelerator branches from the sympathetic, and of the superior and inferior cardiac branches of the vagus, respectively given off from its cervical and thoracic portions. The vagus filaments are known to inhibit the heart's action, and are supposed to subserve sensation, being afferent in this function. Whether or not they contain trophic fibers for the heart is still disputed. Affections of the cardiac branches of the pneumogastric, or neuroses acting through these branches, modify their inhibitory function, or give rise to cardiac sensations, or both.

Tachycardia is undue rapidity of the heart's action. It may be temporary or permanent, and is due to the increased accelerator sympathetic influence or to decreased vagus inhibitory control, or to both acting together. Some individuals have naturally a quick heart. The term

tachycardia is here limited to an acquired rapidity aside from that of exertion, that associated with elevation of body-heat, hemorrhage and weak heart, convalescence from acute illness, etc. It may be temporary or permanent, but usually occurs in attacks or paroxysms in which the patient feels distressed and anxious, often presents flushings and other vasomotor disturbances, and the pulse may be found beating at 120 or even attain a rate of 300 or more a minute. Of this the patient may be unconscious, in which respect it differs from palpitation, an essentially subjective sensation. The attack often terminates rather suddenly, and may be followed by free sweating, copious urination, or even by diarrhea. Nathaniel gives the following distinctions between accelerator irritation and vagus paralysis: Great increase in frequency of pulse, with weak heart-sounds and other disturbances in the pneumogastric area, as aphonia, hoarseness, gastralgia, or cardiac pain, refer to the vagus. Strong heart-sounds and impulse, full peripheral vessels, and vasomotor stasis indicate accelerator disturbance. Whittaker<sup>1</sup> says the increase to 120 beats implies irritation of the sympathetic; from 120 to 180 beats, paralysis of the vagus; above 180, the combined effect of both causes.

Tachycardia may be due (1) to disease of the heart and blood-vessels, (2) to injury of the vagus trunk or nucleus, (3) to toxic causes,—alcohol, nicotine, coffee, and atropin,—(4) to a reflex from any viscus, especially those in the pneumogastric field, and (5) to many nervous, as Graves's disease, hysteria, and neurasthenia. The prognosis and treatment necessarily depend on the causation. The purely nervous forms are difficult to manage, though not of serious import so far as life is concerned.

Cardiac palpitation is sometimes a purely nervous condition, a vagus neurosis. Hysterics and neurasthenics are often much troubled by this rapid heart-action, of which they are perfectly aware. It may come on independently of any assignable cause, as during moments of rest, or even during sleep, though here the influence of some distressing dream can not be excluded. It lacks the anxiety and often associated heart-pain of tachycardia and angina pectoris, but is often attended by tinnitus, vertigo, and a feeling of faintness. All source of cardiac disturbance must be excluded before admitting this form of vagus disturbance.

Unless it is merely symptomatic, the etiology is obscure and its treatment difficult. Sedatives and narcotics, with local applications of heat or cold over the precordium and the administration of diffusible stimulants, may be employed to arrest the attack.

Bradycardia is the opposite of tachycardia. A slow pulse is apparently normal to many individuals and is not associated with any distress or difficulty. Such instances, in which the heart-beats are twenty, thirty, or forty a minute, are on record. The term is here limited to an acquired slow beat that may be permanent, temporary, or paroxysmal. It is sometimes noted in cervical myelitis or injuries to the cord in the

<sup>1</sup> "Twentieth Century Practice," vol. iv.



region. Cerebral conditions marked by pressure, as hemorrhage, hydrocephalus, and tumor, reduce the pulse-rate, and it is often slow in meningitis. The actions of narcotics, biliary poisoning, and anemia only need to be mentioned. Many infective diseases, such as rheumatism, puerperal fever, typhoid, and diphtheria, may produce bradycardia of a persisting sort, or may be followed by it. They probably, at times, set up a vagus neuritis. Organic processes acting on the pneumogastric cardiac inhibitory center, or on its cardiac fibers, may retard the heart by the irritant stimulation of the inhibitory function. Disease of the heart-muscle itself, as in fatty heart and coronary sclerosis, may produce bradycardia, and seems to act by irritation of the terminal filaments of the vagus. The nuclear variety is often associated with arteriosclerotic changes in the medulla.

Bradycardia is usually attended by synaptic disturbances, which has not rarely been mistaken for epilepsy. The paroxysmal form, as in the Stokes-Adams syndrome, is often marked by anxiety, unrest, and distress in the precordial region. Cardiac angina and nausea, or vomiting, may ensue. Convulsions are sometimes noted. When the vagus trunk is affected, other motor symptoms may indicate it, such as aphonia, hoarseness, stridor, and choking. The diagnosis of bradycardia is not difficult, but the heart-beat should never be determined by the radial pulse. The treatment and prognosis depend on the caused condition.

Angina pectoris is an affection of the vagus marked by paroxysms of agonizing pain in the region of the heart, which radiates usually to the left shoulder and arm, and is attended by a sensation of impending death. By some it is called true angina pectoris when dependent, as is usually the case, upon organic heart disease, in distinction from false or pseudo-angina, in which no anatomical changes are discoverable. There is a strong probability that the sympathetic fibers participate in the storm, but the essential element is the vagus neuralgia. The attacks are sometimes induced by exertion or emotion, but may rouse the patient from a sound sleep. Rarely occurring in children, it is much more common in male adults and in the latter half of life, when the arterial changes and cardiac myopathies are commonly found. It may occur independently of such organic changes in neuritic individuals, and is encountered in hysteria, subsiding with that neurosis or suddenly ceasing upon the appearance of other hysterical manifestations.

The pain varies in character and severity. Its neural radiation to the brachial plexus may be replaced by epigastric, diaphragmatic, vesical, and even sciatic pain, ordinarily on the left side. The feeling of impending death, however, is an essential symptom. The pulse may be unchanged even in the presence of the most excruciating pain and the most frightful anxiety, or, rarely, the heart may present tachycardia or bradycardia. The patient is often bathed in perspiration, and in rare cases other pneumogastric conditions, as asthma, laryngeal spasm, or esophagismus, may attend the anginal attack. The attack lasts from a few minutes to an hour, and usually subsides as suddenly as it commenced. The patient feels considerably shaken up and rather languid for a time, but in the intervals of the attacks may be completely free

from all distress aside from that attributable to organic cardiac effects and the apprehension of another attack.

The prognosis depends on the condition of the heart. If it is not organically diseased, the angina does not end fatally, and even cases of organic heart disease seldom die in anginal attacks. The treatment of the attack is antispasmodic. Amyl nitrite and trinitrin hold the first place; chloroform by inhalation, the application of heat or cold to the precordium, whisky, and other similar measures are of some value. In the intervals treatment is directed to the condition of the heart or to the nervous, or to both.

**Gastric Branches.**—The pneumogastric supplies motor branches to the stomach, but only in part innervates the muscular coat of that viscus. Certain gastric movements, such as sallowing and vomiting, undoubtedly depend upon its motor filaments. Vomiting is reflexly produced through its gastric sensory fibers, as well as directly by intracranial disease or irritation of its trunk. Vomiting due to intracranial disease, or that provoked by irritating the vagus trunk and sometimes that due to irritation of its stomach branches by organic disease, is peculiarly rapid, projectile, and, as a rule, unattended by nausea. The vagus probably conveys the sensations of hunger, at least they have been completely destroyed by bilateral lesions of these nerves. Ravenous appetite for food and the peculiar subjective gastric sensations of dyspepsia, organic disease of the stomach, and some neurotic and psychical states are properly attributable to the central or peripheral condition of the pneumogastric nerve.

**Gastralgia, or Gastrodynia.**—Aside from the stomach-pain of organic gastric disease, fermentation, and dyspepsia, there is a neurosis of the stomach marked by sudden pain in this viscus, to which the name nervous gastralgia is given by Ewald. It is not attended by the symptoms of disturbed digestion, and the stomach contents at such times, as well as between the attacks, show nothing chemically irregular. It may alternate with other neuralgias or with migraine, or, rarely, it is associated with an attack of the latter. It is occasionally presented by hysterics, particularly if their attention is centered on the stomach. The gastralgic attack usually comes on promptly and reaches its highest intensity almost at once. The pain is an agonizing, boring, cutting, burning one, and may be localized, diffuse, or in a girdle. It may radiate along the loins or spine. The patients relax the abdominal walls and make deep pressure over the stomach with some relief, though there may be much superficial sensitiveness. Persistent sensitive spots are often found over the lower dorsal vertebrae, between the ribs, and by deep pressure over the abdominal plexuses of the sympathetic. The face is pale, distorted with pain, and covered with sweat. The temperature is not modified. The attacks may be of only a few minutes' duration or may last for hours, and usually terminate rather abruptly, often with the vomiting of mucus or unchanged food. Food is then often actively craved and taken without distress.

These rather rare attacks may very easily be mistaken for gall-stone colic, acute indigestion, gastritis, gastric ulcer, and a host of other abdom-



inal conditions which must be excluded to make the diagnosis possible. In locomotor ataxia they furnish the gastric crisis due in this disease to irritation of the pneumogastric nucleus in the medulla, where any other localized lesion may provoke them. They are, as a rule, associated with constipation. If due to organic nervous disease, the treatment and prognosis correspond. The same is true in hysterical cases. Otherwise the general upbuilding indicated in all neuralgic conditions must be attempted.

**Rumination**, or *oryxism*, is occasionally observed in man. The food is voluntarily regurgitated, remasticated, and again swallowed. It occurs in neurasthenia, hysteria, epilepsy, and idiots. Usually the rumination begins a short time after the meal and lasts for half an hour. Such patients often insist that they are unable to control the habit, but, except in idiots, do so, at least to some extent, in the presence of strangers.

**Nervous dyspepsia**, a condition set up by Leube as a gastric neurosis, is claimed by Ewald, and others equally competent, to be but a local manifestation of neurasthenia. Ewald points out that there is no peptic deficiency, and even Leube based his diagnosis largely on the fact that a meal is thoroughly digested and the stomach empty within the alleged normal limit of seven hours. The digestive difficulties of neurasthenia will be mentioned under that caption.

## CHAPTER IX.

### DISEASES OF THE SPINAL PORTION OF THE ACCESSORY NERVE.

**Anatomical Considerations.**—The external portion or spinal part of the accessory nerve is, properly speaking, not a cranial nerve at all. Arising by a series of roots from multipolar cells in the anterior spinal gray matter throughout the cervical cord as low as the sixth segment, it is finally gathered into a bundle or trunk. This passes upward through the foramen magnum and joins the true accessory portion. It then passes with it through the jugular foramen, when it finally separates to be distributed to the sternomastoid, which it entirely, and to the trapezius muscle, which it partly, furnishes with motor and trophic control. The trapezius is also supplied by numerous spinal branches, particularly in its lower portion, and only depends on the spinal accessory in its upper half, though Bailey<sup>1</sup> reports a case in which division of the spinal accessory paralyzed the trapezius and sternomastoid completely and produced a serious disability. This nerve may be centrally or peripherally affected, and the result is spasmic or paralytic as the lesion is irritative or destructive.

**Accessory Spasm.**—*Spasmodic Torticollis*, *Spasmodic Wryneck*.—Irritative lesions of the spinal accessory produce characteristic action and

<sup>1</sup> Ann. of Surg., 27 May, 1901.

prominence in the muscles it supplies. By the sternomastoid the face is carried forward, turned toward the opposite side, and tilted upward. The trapezius draws the head backward and to the same side, lifts the shoulder, and shortens the distance between the occiput and scapula. At the same time the scapula is drawn nearer the vertebral spines as well as upward. Both sternals acting together bring the head forward and downward. Both trapezii carry the head backward and the face upward. Acting alternately they turn the head from side to side with slight rotation. The spasm may be clonic or tonic, may involve one or both sides, and may be limited to the sternals or the trapezii. A tendency to spread to other muscles of allied function is often observed. As a matter of fact, in most cases of wryneck more muscles are involved than those supplied by the accessory nerves. Thus the splenius and the small rotators are frequently associated in the spasm, just as they are related functionally. The large muscles named are, however, the ones most at fault and the ones that impress a given case with its distinctive movement or attitude. The trapezius is less often clonically involved than the sternomastoid, but is as frequently affected by tonic spasms. In some cases clonic torticollis is a localized tic, or it may be a portion of a more widely distributed tic such as that arising in the face and gradually involving the neck, shoulder, and arm. In a mild form it furnishes the habit spasm that follows chorea sometimes or is picked up by pubescent youths. We may also have a mental torticollis as described by Rouquies,<sup>1</sup> in which the patient can not voluntarily restore the head to its natural position, but turns it readily in all directions if allowed to press it against any object, or to apply an insignificant amount of manual assistance. The spasm is here the result of a mental obsession. The nodding spasm of hysteria arises in the same way, and in both the disorder must be referred to the cortex or at least to the higher mental levels. In this connection the subject of Tics in Part VII. should be read. Cerebral tumors, meningitis, focal softenings, and disease of the medulla may underlie the spasm. Cervical spondylitis and meningitis usually produce tonic spasms. The action of cold, cervical adenitis, and violent wrenching of the neck may peripherally excite them. Torticollis has been reflexly produced by intestinal worms.

Treatment is often highly unsatisfactory. The variety due to cold usually subsides spontaneously or may be benefited by local applications and antispasmodics. The hysterical form may suddenly cease. The cases dependent upon organic lesions are often beyond reach. Sedatives like camphor, opium, and hyoscin only give temporary relief and often upset the stomach or, unfortunately, lead to their habitual use. When the spasm is severe, and especially if confined to one side, wide resection of the spinal accessory nerve before its entrance into the sternomastoid is advised, other means having failed. A tic or habit is thus likely to be benefited for a time, but is also likely to reappear in some adjoining area. Stretching the nerve is almost sure to be followed by a relapse of the tic as the nerve recovers, as is the case also if the divided nerve unites.

<sup>1</sup> *Thèse de Paris.*



The upper spinal nerves to the small rotators must in some cases also be divided to completely quiet the spasm.

Children sometimes present a permanent wryneck of an entirely different character. Owing to traction on the head and twisting of the



Fig. 6L.—Tollosity in wryneck soon after division of left spinal accessory.

neck in labor, or even in unaided labor, the sternomastoid may be injured, and subsequent contracture shortens it. The result is a firm, fibrous band that holds the face to the opposite side. Inflammatory injury to the muscle in adults may cause the same thing, and in both cases it can be remedied only by thorough division of the shortened tissue.

**Accessory Paralysis.**—Cerebral disease very exceptionally results in complete and permanent loss of power in the spinal accessory area. Like those of other bilateral movements, these muscles seem fully represented in both hemispheres. As a part of nuclear disease, as in progressive muscular atrophy, the spinal centers are often involved, with a corresponding loss of power and nutrition. By meningitis the trunks of one or both nerves may be implicated at the foramen magnum. The hypoglossal is then likely to suffer with them, and the true accessory fibers are usually also affected. Outside the skull the spinal portion of the accessory is sometimes involved in wounds, operations, deep-seated tumors, vertebral caries, cervical adenopathy, and by neuritis. The unilateral symptoms are loss of power and wasting in the related muscles. This involves the sternomastoid almost entirely and the trapezius only in its upper portion. The head is not so readily and strongly turned to the opposite



Fig. 6C.—Wryneck, stiffness, lengthening of neck, and rotation of head after division of left spinal accessory.

The unilateral symptoms are loss of power and wasting in the related muscles. This involves the sternomastoid almost entirely and the trapezius only in its upper portion. The head is not so readily and strongly turned to the opposite

side, the shoulder droops slightly, and the extension power of the arm is lessened. The scapula moves outward, especially its upper inner angle, and stands out from the ribs. The curved line from mastoid to acromion becomes depressed and even angular upon efforts at deep inspiration or in extending both arms against resistance. Subsequent contracture in the unopposed muscles of the sound side may turn the face to the primarily affected side.

In the bilateral form, usually due to meningitis or vertebral caries, the head is held insecurely and readily falls forward or backward as the trapezius or sternals are most affected. Injury to the spinal accessory in the posterior triangles of the neck after it has passed through the sternomastoid only affects the trapezius. The treatment is that of the causal condition in the given case. In neuritis electrical stimulation is indicated, and nerve-suture would be required in the cases where the nerve had been divided.

## CHAPTER X.

### DISEASES OF THE HYPOGLOSSAL NERVE.

**Anatomical Considerations.**—The twelfth cranial nerve is a purely motor nerve for the muscles of the tongue and subserves their nutrition. Its cortical centers adjoin those for the lips in the lower portion of the ascending frontal gyrus. Its lower neuron arises in the hypoglossal nucleus under the floor of the fourth ventricle. This nucleus is close to the median line, and is made up of large cells analogous to those in the anterior spinal horn of gray matter. The nerve-trunk arises by a number of roots in the groove outside of the olivary body, and, passing forward, leaves the skull by the anterior condylar foramen of the occipital bone. It has a connection with the pneumogastric, which it accompanies a short distance, and receives a branch from the upper spinal nerves. This branch eventually innervates the depressors of the larynx bone, and is not affected by injury to the hypoglossal nucleus. The hypoglossus may be injured or diseased in any part of its course from the cortex to the peripheral branches, and manifests such injury by spasm, paresis, and paralysis, as the causal state is irritant, inhibitory, or destructive. In addition, injury at or below the nucleus entails atrophy of the muscular fibers of the tongue. The mucous portion of the tongue is supplied by the trifacial.

**Hypoglossal Spasm.**—The tongue is usually involved in epileptic convulsions, and is frequently bitten in consequence of having been thrust between the teeth by the action of the geniohyoid muscles and lacerated by the spasmodic action of the mastoidors. This may occur in very slight attacks, when no convulsive action in the extremities is observed. At an early stage of hysterical convulsions the tongue is usually protruded violently, but is only bitten in extremely rare cases. This lingual spasm may comprise the entire motor disturbance in some hysterical cases. In chorea the tongue is usually involved in the muscular twitchings that mark the disease, and is often the seat of vigorous



choreic movement, in which it may be protruded and even slightly bitten. The speech difficulties of this disease are due in part to the lingual spasm. From forced occlusion, as in public speakers, it is sometimes the seat of a neuræic manifestation similar to writers' cramp, a condition termed *aphthongia*. Slight spasm also occurs in stammering and in stuttering. Lingual spasm has been reflexly dependent upon decayed teeth and cured by their removal.

Cortical irritation has, in rare instances, given rise to lingual spasms that practically constitute Jacksonian epileptic attacks. The lips and face usually participate in the spasm, as the contiguity of their centers would lead one to expect. In some of these cases the initial convulsion has been a generalized fit; in others such attacks have followed. It is conceivable that nuclear irritation might give rise to similar manifestations, but post-mortem data for this belief are lacking. The treatment would be that of the general condition underlying the local manifestation.

**Hypoglossal Paralysis.**—The hypoglossal nerve may be paralyzed by injury at any point in its course. In rare cases a limited cortical lesion has produced paralysis of the opposite side of the tongue. More widespread cortical lesions and lesions in the supranuclear motor tract usually produce a one-sided paresis or paralysis of the tongue, which, on protrusion, deviates toward the paralyzed side,—that is, away from the lesion. This is due to the action of the unaffected and properly acting genioglossus of the sound side. Nuclear disease usually produces bilateral palsy of the tongue, which lies motionless in the floor of the mouth, and, after a few days, rapidly wastes. A few cases of one-sided nuclear disease are recorded. In bulbar palsy, syringomyelia, and tabes it is thus affected in exceptional cases. Other cranial-nerve nuclei usually suffer at the same time, and the resulting symptoms enable one to localize the disease. The fibrillar twitchings in the tremulous tongue of parietic dementia and some of the stammering of this malady are due to the nuclear and cortical invasion of the disease. The root of the nerve in its passage through the medulla is sometimes damaged by a local lesion, which of necessity interferes with the pyramidal tracts for the opposite side of the body and produces a crossed paralysis of the tongue and limbs. The tongue, in such an instance, would deviate from the paralyzed side of the body and toward the side of the lesion. Such a crossed palsy, due to disease of the olivary body, has been reported.<sup>1</sup> Basilar processes, like meningitis and fractures, or bone disease involving the condylar head, may injure the nerve in its intracranial course. Outside of the skull it is vulnerable to penetrating wounds, or may be implicated in deep abscesses, as from caries of the upper vertebrae. Here its neighbors, the spinal accessory and the pneumogastric, are likely to be involved at the same time. It may be affected by a neuritis as in a case reported by Pinski,<sup>2</sup> who was able from the literature to collect 40 cases of isolated hypoglossal paralysis due to peripheral disease or injury.

In unilateral hypoglossal paralysis due to damage of the nerve at or below the nucleus, the paralyzed side of the tongue shows a marked

<sup>1</sup> Gorkowsky, "Société Iconographique de Neurologie," No. 3, 1905.

<sup>2</sup> "Neurolog. Centralbl.," Aug., 1903.

loss of volume. This does not follow supranuclear lesions. The mucosa covering, on the other hand, not being deprived of its trophic supply, is thrown into marked and apparently excessive folds. Taste and sensation are not impaired. Mastication on the paralyzed side of the tongue is not well performed, as the patient finds difficulty in placing and maintaining the food between the teeth. When protruded, the tongue curves sharply to the wasted side, but within the mouth motions toward the palsied side are wanting. In the bilateral form speech is much affected, as nearly all consonant sounds depend in some degree upon the position of the tongue. Swallowing is also difficult, as the bolus or fluid is not readily carried backward into the pharynx and mastication is greatly impeded.

Treatment is directed to the causal condition, aside from which it is practically hopeless.

## CHAPTER XL

### MULTIPLE PARALYSES OF CRANIAL NERVES.

In discussing the cranial nerves separately, frequent reference has been made to their association in diseased processes. Sometimes the limitation of this association is of diagnostic importance, as when the loss of hearing with facial palsy denotes a lesion of the seventh and eighth nerves in their parallel intracranial course or within the internal meatus auditorius. In meningitis and other basilar processes a number of cranial nerves are usually implicated at once, and such multiple paralyzes of cranial nerves become symptomatically highly important. The close juxtaposition of the cranial nuclei has been repeatedly pointed out as giving rise to associated palsies in bulbar disease, such as tumor, hemorrhage, and limited basilar thrombosis. In their cortical folds and their supranuclear motor paths a number of the cranial nerves may also be injured at once by transection or disease. Hypoglossal palsy with hemiplegia has just been called to attention, as well as the association of hypoglossal and facial spasm due to the cortical relations of their centers. These multiple cranial palsies are principally of symptomatic interest and value, but there are groups of nuclear palsies of the cranial nerves that present well-marked clinical forms of disease. Their pathological anatomy is largely confined to the upward extension of the spinal gray that furnishes the chain of cranial-nerve nuclei reaching from the lower angle of the floor of the fourth ventricle to the posterior portion of the third. Anatomically and clinically these diseases fall into two forms: First, those of the upper group, primarily implicating the nuclei of the third, fourth, and sixth nerves (see Fig. 36), and, second, those first implicating the remaining lower cranial nuclei. As these diseases are sometimes inflammatory in character and analogous to poliomyelitis, they have come to be known respectively as *polio-encephalitis superior* and *polio-encephalitis inferior*. More commonly the process is one of degeneration. The analogy, or in some cases the identity, of the disease of the nuclei of the bulb with that of the anterior gray of the cord is now generally conceded. It is the same process arising in different localities, but affecting homologous elements



and producing results similar in kind. Transition and combination cases of every variety are of record. Commencing in the upper nuclei of the ocular nerves, the lower bulbar levels may be progressively involved, and later spinal features may be added; or, beginning in the bulbar nuclei, an upper extension may induce progressive ophthalmoplegia. In the same way that the spinal type of the disease may be acute, subacute, and chronic, we have an acute, subacute, and chronic poly-encephalitis superior and inferior, and forms combining both.

**Polio-encephalitis superior chronica** will be considered first, as it presents the most complete and well-defined picture. It is also called *progressive ophthalmoplegia* and *chronic nuclear ocular paralysis*. It is characterized by the gradual loss of power in the external and internal muscular apparatus of the eyeball, one set of muscles after another being gradually involved. Usually bilateral, it may be confined to one eye for a period even of years and then invade the other. As a rule progressive, it may come to a permanent standstill at any time, or advance intermittently.

**Etiology.**—Poly-encephalitis superior has in so many cases followed infections and toxic states that it has with reason been attributed in some cases to pneumonia, grip, diphtheria, and syphilis; to lead, sulphid of carbon, carbonic-oxid gas, sulphuric acid, and alcohol. A careful search for syphilis by blood and spinal fluid tests indicates a greater relationship to this infection than was formerly supposed to be the case. These toxic agents, however, are more common in the causation of the acute variety, which may terminate by becoming chronic. It may be secondary to or a part of tabes dorsalis, and the characteristic Argyll-Robertson pupil of that disease is a part of it. It is similarly related to posterolateral spinal sclerosis, general paresis, and insular sclerosis. It may be an upward extension of identical disease in the cord or bulb. It is more frequent in men than in women, and while it may appear at any age, it is principally a disease of adult life. In some cases there seems to be a teratological defect, congenitally manifest or appearing during the active periods of growth.

**Pathological Anatomy.**—The lesions are variable in extent. They involve the upper cranial-nerve nuclei on one or both sides, including those of the third, fourth, and sixth nerves, wholly or in part. When partial, the iridosiliary nucleus of the third and its adjoining nucleus for the elevator of the lid most frequently escape or are only partly destroyed. The corresponding nerve-trunks and muscles are degenerated and atrophied. In addition are encountered cerebral, bulbar, and spinal conditions, of which poly-encephalitis superior sometimes is but a part. Microscopically, especially in the early stage, some hyperemia is found about the nuclei. Later the cells of these nuclei lose their prolongations, are diminished in size, present vacuoles and pigmentary degeneration. Some completely disappear, and in cases of long standing but a few atrophied remnants remain. Slight capillary hemorrhages or their traces can at times be determined. Some leukocytic infiltration occurs about the vessels or in the foci of the disease, and minute sclerotic patches are found. Rarely the lining of the Sylvian aqueduct is thickened.

**Symptoms.**—In the fully-developed cases the faces, classically de-

scribed to Hutchinson, and recognized by Brunner, von Graefe, Charcot, and others, is most striking. The drooping lids partly covering the eyes cause the patient to hold the head back and strain with the frontalis to overcome the partial ptosis. The brow is thrown into deep transverse wrinkles. The immobile eyes and inactive pupils are like those of glass in a mask of wax. They usually deviate outward, as the sixth nucleus is often spared at first; but any squint may in various cases be present or the visual axes may conform. The ptosis is usually less marked after a night's rest; and if the ocular muscles still retain a slight degree of activity, this, too, is best in the morning. Diplopia is rarely mentioned by these patients, which Blane attributes to the slowness with which the deviation develops, thereby allowing the brain to neglect one image. In partial cases the ocular conditions may be unilateral or they may be only functionally distributed and bilateral or unilateral. Thus the iris may react for light or accommodation, or both. The sphincter may at first escape or first be involved, and innumerable combinations and varieties have been encountered as one or many nuclei were partially or completely affected.

The general health of the patient may leave little to desire or he may present the somatic conditions and functional disturbances that belong to the associated diseases already mentioned. In some instances involvement of the fifth nerve, probably through its descending root, has given rise to paresthesia or anesthesia in the face.

**Course.**—The evolution of the disease is one of its most distinctive features. Commencing with a ptosis or a squint, the other features of the disease are gradually added during, perhaps, several years. Stationary periods of long duration—twenty-five years in a case of Strimpell—may intervene, and finally the disease takes up its progressive course, perhaps without appreciable cause. Extensions of the process to the bulbar region may occur, and we have bulbar palsy or poli-encephalitis inferior added and the prognosis for life becomes much darkened. The lesions may not stop at the bulb, but may invade the cord, producing a progressive spinal muscular atrophy of any one of the various types of that disease.

The diagnosis depends largely upon the evolution of the disease and its progressive course. When thoroughly developed its remarkable facies is not easily mistaken. The acute form, and hemorrhage, inflammation, and softening, give a history of rapid onset and promptly reach their maximum stage. In multiple neuritis, especially the alcoholic and diphtheric varieties, confusion is likely to arise, but we may generally be guided by the condition of the extremities and the wide diffusion of the disease. Even then we can not absolutely exclude the spinal nuclei, which may become the seat of a chronic atrophic process. An orbital cause may give rise to a partial and increasing ophthalmoplegia, but proptotic symptoms, exophthalmos, and optic atrophy will finally distinguish it. Basilar processes and bulbar growths, aside from the distinctive symptoms to which they give rise, usually implicate so many nerves and the pyramidal tracts that they can be easily separated.

Treatment turns practically upon the presence or suspicion of syphilis, which should be vigorously managed. Other toxic possibi-



ties, such as lead-poisoning, will require appropriate intervention. Strychnin does temporary good sometimes. Electricity is futile, as it can not be applied to the faulty muscles. In the majority of cases the physician is helpless.

**Acute and Subacute Polio-encephalitis Superior.**—The acute form is very rare. Like its congener, acute poliomyelitis, it may be purely an infectious disease, and has been frequently noted in recent epidemics of the spinal disease. It may follow many acute infections, as mentioned in the etiology of the chronic form, especially diphtheria, pneumonia, and influenza. The subacute form, while in some cases attributable to the above causes, is more often due to syphilis and toxic agents, like lead and the carbon gases. The onset is sudden or very acute, and the disease may reach a fatal termination in a few days or a week, usually from implication of the bulbar centers. The nuclei in several such cases have presented no postmortem changes that could be discovered. Bissaud<sup>1</sup> supposes that the infection has overwhelmed the nuclei before histological traces discoverable by our present means of investigation were developed. The similarity to Landry's paralysis in this regard is striking, and the diseases may be essentially the same, varying only in localization. In other cases well-marked inflammatory changes have been discerned. The prognosis is very grave, especially in children. Some cases come to a standstill; others develop into the chronic variety.

The treatment should be directed to securing perfect rest and the removal of any source of infection or intoxication and its eradication from the system before important changes occur.

**Polio-encephalitis inferior chronica** is the term employed by Wernicke to denominate the disease first described by Dumesnil, later by Duchenne, as progressive paralysis of the tongue, palate, and lips. It is also known as *laryngopharyngeal* or *pharyngeal paralysis* (Leyden), as *progressive bulbar paralysis* (Wiedemann), as *paralysis of the bulbar nuclei* (Kussmaul), and as *progressive bulbar palsy*. It consists in a progressive paralysis of the lips, tongue, pharynx, and larynx, with wasting of their muscles, and usually terminates in death from pneumogastric palsy. The disease process is limited to the nuclei and lower neurons of the seventh, ninth, tenth, eleventh, twelfth, and the motor portion of the fifth cranial pairs. It is identically the same in character with progressive spinal muscular atrophy, with which it is often associated. In this connection the reader is referred to the description of the spinal type of progressive muscular atrophy in Part V of this work.

**Etiology.**—Males and females are about equally susceptible to this disease. Though it has rarely been observed in childhood and may be a congenital or hereditary deficiency, it usually appears after the age of thirty. Occasionally, and then ordinarily as a part of a more extensive spinal atrophy, or in association with superior polio-encephalitis, it has appeared in successive generations or in more than one member of the same family. These family cases usually develop in early life. It has

<sup>1</sup> *Lecours*, 1895.

been attributed to exposure to cold; to the overuse of the mouth-muscles, as in players of wind-instruments and glass-blowers; to syphilis, to Bright's disease, and other wasting maladies. It may be a part of amyotrophic lateral sclerosis. It has appeared in tabes dorsalis, insular sclerosis, syringomyelia, and followed descending degeneration of the pyramidal tracts after cerebral lesions. Knowledge of the rôle of bromin in these chronic disturbances is widening. Progressive bulbar palsy has been known to follow lead intoxication and diphtheric poisoning. Often the causation is entirely obscure.

**Morbid Anatomy.**—The lesion is limited to the nuclear cells in the lower half of the bulb. It is practically symmetrical. The hypoglossal nucleus is most severely affected, as a rule. The nuclei of the true spinal accessory, the facial, the motor portion of the trifacial, and the pneumogastric are invaded with decreasing intensity and frequency. A degenerative process is found in the nerve-trunks whose nuclei are affected and their muscular terminations waste. The muscle-fibers



Fig. 51.—Case of bulbar palsy. 1, Photograph taken shortly before first symptoms were noted; 2, photograph taken four years later, a few weeks before death.

themselves their corresponding degeneration and atrophic conditions. The minute anatomy is the same as that in progressive ophtalmoplegia or that of progressive spinal muscular atrophy, to which the reader, bearing in mind the special location of this disease, is referred. The organic changes of associated atrophies and sclerosis or of primary conditions, such as tabes, syringomyelia, insular sclerosis, and descending cerebral degenerations are at times encountered.

**Symptoms.**—The symptoms begin insidiously and progress slowly. The tongue is usually the first affected. This is manifest in a thickened pronunciation, particularly of the letters which require definite lingual movements. The linguodentals and linguopalatals (see table, p. 97) and the vowel "c" and later on the explosive labial sounds are lost. Finally speech is reduced to unmodified laryngeal noises that are quite unintelligible. When the disease is only slightly developed, by an effort the patient can often enunciate clearly and the embarrassment may be noticed only in ordinary inattentive conversation. The tongue also progressively loses its muscular strength and range of motion until it



lies a flabby, inert, rugose, atrophic mass on the floor of the mouth. It loses its function of maintaining the food between the teeth during mastication and of carrying the bolus backward and thrusting it into the pharynx in efforts at swallowing. It can no longer be protruded, turned to either side, rolled up, or hollowed into a gutter. In most of the cases it notably wastes, but as the atrophy is confined to the muscular portion of the tongue, the dermal covering appears too large and may even suggest epidermal hypertrophy.

The lips are affected shortly after the tongue, and this adds to the speech difficulty by rendering the pronunciation of the vowels "a" and "u" uncertain or impossible. The labial consonants are lost as above indicated, and little besides the sibilant "s" and the open vowel "a" remain. The orbicularis oris is usually the first labial muscle invaded, but all of the labial group are eventually paralyzed. Their nuclear association with the hypoglossus and their functional relationship will be recalled. At first whistling and blowing effects are weakened, but finally the mouth hangs loosely, the lower lip drooping away from the teeth, and all voluntary movements are lost. This, with the action and eventual contracture of the zygomatics, serves to greatly accentuate the nasolabial furrows and imparts a demented appearance to the lower portion of the face.

Out of the drooping, open mouth saliva drips, requiring the constant use of a napkin or handkerchief. The quantity is sometimes enormous and always appears greater than normal. When the masseters and pterygoids are involved, which is usually at a late stage, but may be an initial condition, mastication is feeble or impossible. Finally, their complete paralysis allows the mandible to hang loosely, increases the opening of the drooping mouth and the salivary overflow. The jaw-jerk is abolished except in those cases where spastic symptoms elsewhere point to the association of the pyramidal tracts in the lesion. It is then increased. The lips usually show marked atrophy and are sensibly thinned. This is sometimes obscured by the fatty deposit, but in the final stages is practically a constant condition.

The palate follows the lips in order of involvement in a majority of cases. Its loss of muscular tone is manifested by the nasal voice tones, which also adds to the lack of clearness in the pronunciation of the linguo-palatale, and it turns "p" and "b" into the nasal resonant "m." When the palate is fully palsied it hangs loosely in the pharynx without reflex action or voluntary movement. As it can no longer shut off the nasal



Fig. 34.—Mouth in bulbar palsy. Voluntary mouth-closing and all thrust to prevent the tongue, which lies inert, and droop down in the floor of the mouth.

spaces, fluids often regurgitate through the nose, and even food masses may be forced into the nasal fossae.

When the pharynx is involved, the difficulties of swallowing reach their maximum. Food is now prone to enter the respiratory tract and a violent, fatiguing, and alarming cough is often produced. The danger of pulmonary engorgement, aspiration pneumonia, and heart-failure is intensified by the pneumogastric weakness that is often present. Alimantation becomes so difficult that the esophageal tube must be used or the patient must be fed by the bowel. To these difficulties is added, sooner or later, a paralysis of the larynx. The adductors are usually most affected and the glottis stands wide open, serving neither the purposes of phonation nor protection to the trachea against the entrance of foreign material. In very rare cases unilateral or bilateral abductor palsy is found, and in the latter case inspiratory stridor indicates the dangerous respiratory difficulty. Both pharyngeal and laryngeal reflexes are abolished. The voice is extinguished. Failing respiratory and cardiac actions lead to a final termination, which may come suddenly at any period of the disease. It is often induced by aspiration pneumonia or caused by suffocation due to blocking of the respiratory passage by a mass of food.

The pulse is likely to become frequent, weak, and irregular. The agonal attacks are not infrequent. Senescence may occur and prove fatal. The pneumogastric involvement further shows itself in a feebleness of respiration, so that coughing and other active expiratory efforts become almost impossible, adding greatly to the danger of choking and to the general discomfort of the patient. It is somewhat remarkable that polyuria and glycosuria are seldom encountered. The controlling centers in the bulb are in close proximity to those invaded by this disease. It will be noticed that the nuclei selected are purely motor and trophic, and that the disease spreads not so much by contiguity as along lines of associated function. This is one of the distinctive habits of the disease, and serves here, as in other progressive maladies of the cerebro-spinal axis, to draw earnest attention to this factor in the study of every case.

The electrical examination of the atrophic muscles presents considerable difficulty, excepting in the lower face and the masseters. The changes found are practically a quantitative reduction to all currents as fiber after fiber disappears. The reaction of degeneration is wanting. The reflexes are diminished in a degree proportional to the atrophy, excepting in those cases in which the disease early involves the pyramidal tracts. Sensation is not markedly affected, nor is the sense of taste notably disturbed in pure cases. The temperature remains unimpaired throughout, save by intercurrent accidents.

**Course.**—The disease is one of insidious onset and its steadily progressive course is characteristic. In a few exceptional cases the progress of the disease presents intermissions, but remissions are practically unknown. The duration of the malady from inception to fatal termination may be roughly stated as from one to five years. Leyden reports one case of seven years' duration, but, on the other hand, intercurrent maladies and



the suffocative, cardiac, and pulmonary accidents to which the disease lays the patient liable, may cut life short at any moment. The increasing feebleness and malnutrition at the same time add to the gravity of the situation. As above indicated, the disease first manifests itself in the tongue and progressively invades the lips, pharynx, palate, masticators, and larynx. This is a usual sequence, but not a necessary one. Any modification of it may be presented. The encroachments of related nuclear disease at lower and higher levels give rise to different trains of symptoms, which, however, in their full development furnish very similar pictures. The course pursued in any given case can be understood by reference to the anatomical and especially to the functional relationship of the bulbar nuclei. The occurrence of a bronchitis, bronchopneumonia, angina pectoris, or of suffocative attacks is often of fatal import.

**Diagnosis.**—The diagnosis of a well-developed and unmixed case presents little difficulty. The course of the disease is of the first importance. The facies can hardly be mistaken. Palsy of both facial nerves gives rise to feebleness of the lips, but the upper part of the face does not escape and glossopharyngeal symptoms are lacking. Diptheritic palatal palsy may raise a doubt unless the clinical history of the infection is available. In this condition the lips and tongue escape; the onset is somewhat abrupt, and the usual course is toward recovery. Great difficulty may be presented in cases of multiple neuritis, but in these we have sensory disturbances, the reaction of degeneration, and marked symptoms in the extremities. When secondary to tubercle, insular sclerosis, syringomyelia, and amyotrophic lateral sclerosis, the highly characteristic symptoms of these various diseases are prominent. As an extension process from the cord upward, or from the ocular nuclei downward, its development is preceded by the well-marked evidence of these prior states, which persist and increase during the evolution of the bulbar paralysis. The greatest diagnostic difficulty is presented by cases of the pseudobulbar paralyses.

**Treatment.**—In pure polio-encephalitis inferior chronic the prognosis is fatal. Curative treatment is, therefore, out of the question, but much can be done to alleviate the distressing condition of the patient and to obviate the laryngeal, pulmonary, and nutritive dangers that threaten him with suffocation, asphyxiation, pneumonia, and inanition. The hypersecretion of saliva may be checked by atropin, which also furnishes a reliable heart-stimulant. The stomach-tube and rectal alimentation are our means to avoid strangulation and to secure nutrition. Recourse to tracheotomy may be had in absolute laryngeal paralysis. Electricity is of use in exercising the muscles of the face, tongue, and gullet. The faradic current is sufficient. The large, indifferent electrode may be placed on the back of the neck, and a smaller, active electrode is then brought into contact with the lips, masseters, and tongue. By placing it over the pons an adam's swallowing efforts are induced. Care must be exercised not to fatigue muscles already wasted. Energetic courses of silver, ergot, phosphorus, zinc, picrotoxin, and mercury, except mercury and arsenic in specific cases, are mentioned

only to condemn them, and anything else that pulls down the waning strength of the patient must be avoided. Tonics, rest, and strengthening measures are of value.

**Acute bulbar palsy**, or *acute bulbar myelitis*, is due to the same infections that set up acute myelitis. It may furnish the terminal stage of the chronic form, or it may result from an upward extension of a condensation. The symptoms with which we are familiar in the chronic variety are rapidly evolved and often associated with febrile disturbances, headache, and somnolence. As the disease gains the pneumo-gastric nuclei, death becomes imminent and results through respiratory failure.

**Combined Forms of Polio-encephalitis.**—The various combinations of chronic superior and inferior polio-encephalitis by extension have been alluded to in describing them separately. In some very rare cases the nuclear invasion falls upon upper and lower cranial nuclei practically at once. The resulting picture is an aggregate of the simpler ones. From their vital nature, the presence of pneumogastric symptoms dominates the outlook.

**Pseudobulbar Paralysis.**—These are (1) organic and (2) asthenic. The organic variety is due to more or less symmetrical lesions involving the posterior group of cranial nerves at any point from their cortical centers to their peripheral trunks. We distinguish a *vascular form* due to bilateral cortical or subcortical vascular lesions. The onset is abrupt, and usually developed in two stages. A hemiplegic or apoplectic case presents a second stroke, this time from a lesion in the second hemisphere, and the bulbar palsy is at once established or completed. It is only very rarely limited to the parts definitely elected by true bulbar palsy, does not present the atrophy or degenerative reactions, and the reflexes are retained or exaggerated. A *embolic form* follows acute bulbar myelitis and hemorrhage into or softening of the bulb. These are rare affections of sudden onset. The lesion does not spare the motor tracts for the limbs, and other bulbar functions do not escape. Tumor may similarly produce a pseudobulbar palsy, but the distinctive symptoms of an intracranial growth—headaches, vomiting, vertigo, and papillitis—are added. A *vascular form* is occasioned by tumors of the base and basilar meningitis, especially of the syphilitic variety. A pseudobulbar paralysis of this form is among the greatest mimics, for obvious anatomical reasons. Finally we have a *neuritic form*. This is usually only a part of a more widely distributed or multiple neuritis.

**Asthenic Bulbar Paralysis (*Myasthenia Gravis*).**—This disorder, as described by Strümpell,<sup>1</sup> may mimic chronic polio-encephalitis inferior very closely, but is never so definitely limited to the cranial nerves. Previous to his communication a similar case was reported by Jolly,<sup>2</sup> under the title of "*Myasthenia Gravis Pseudoparalytica*." Since that time cases have been reported by Murri,<sup>3</sup> Pincus,<sup>4</sup> Bruns,<sup>5</sup> Collins,<sup>6</sup> Hallervorden,<sup>7</sup> Kojewnikoff,<sup>8</sup> and many others.

<sup>1</sup> "Deut. Zeit. f. Nervenheilk.," Bd. 8.

<sup>2</sup> "Berlin. Klin. Wochens.," Jan. 7, 1885.

<sup>3</sup> "Wien. Jahrbuch f. Psychiat.," vol. xii.

<sup>4</sup> "Internat. Med. Mag.," April, 1896.

<sup>5</sup> "Deut. Zeit. f. Nervenheilk.," Nov., 1896.

<sup>6</sup> "Politiason," vol. 3, 1885.

<sup>7</sup> "Schmidt's Jahrbuch," 1896.

<sup>8</sup> "Archiv f. Psychiatrie," vol. xxv.



The condition in most instances has terminated fatally; but no changes in the bulbar nuclei were discovered. Apparently the deficit or toxic effect had not reached a degree sufficiently intense to produce cell-changes that were observable under ordinary methods of examination. In a case reported by Vidal and Maresco,<sup>1</sup> disintegration of the chromophilic elements was demonstrated by the Nissl and Marchi methods. Goldfuss<sup>2</sup> found widespread and decided changes in the muscles. This case and one reported by Long and Wicki presented preceding chronic pulmonary septic conditions, and this association is probably not uncommon. The sepsis of phthisis pulmonalis may run into a grave myasthenic condition, as I have seen in one case which presented all the characteristic bulbar features. Laquer and Weigert have also noted widespread changes in the muscles apparently secondary to thymus disease. Laquer reports a case followed by progressive spinal muscular atrophy. Senator<sup>3</sup> suggests a relationship to various depraved blood states. Bernak refers to a case of associated Graves' disease. Feinberg reports a case in which stercoræmia was present and the septic symptoms relieved upon its relief. A persistent and enlarged thymus gland has been found so frequently, generally in association with widespread intramuscular infiltration of cells of a lymphoid character, that more than a casual relation seems to be implied between the myasthenia and the glandular state. Von Kely,<sup>4</sup> after a study of 134 cases with 42 autopsies collected from the literature, concludes that a neuropathic heredity is practically the only common antecedent, that the nervous system is intact, and that the muscles are the seat of the disease, which is the result of some auto-intoxication. Csiky<sup>5</sup> finds that the reported cases of this lymphoid state may be divided (1) into such as present a primary tumor in some part of the body and (2) those which have no such association. The second group is much the larger. Bernard has found the lymphoid infiltration in the heart, liver, adrenals, and thyroid. An intimate relation of the disease to exophthalmic goiter and associated disorders of the glands of internal secretion is emphasized by Stern.<sup>6</sup>

The paralysis, which comes on more or less insidiously, especially involves the tongue, lips, and pharynx, but the eyes and extremities are also affected to a certain degree, and sometimes very decidedly, and weakness of the muscles of the neck is a notable symptom. Ptosis is commonly encountered early. Usually, indeed, the myasthenia is general. Filiciliary tritling is wanting, and the reflexes are not disturbed,<sup>7</sup> excepting that, if repeatedly elicited, they tend to fail through the induced muscular fatigue. Electrical responses are only modified by the fatigue induced by their repetition, presenting the myasthenic reaction, especially to the faradic current. It is found, as in bulbar paralysis, that rest seems to improve the paralytic features, but that the muscles

<sup>1</sup> "Presse méd.", April 14, 1897.

<sup>2</sup> "Neurol. Centralbl.," Feb. 1, 1902.

<sup>3</sup> "Berlin. klin. Wochens.," 1896.

<sup>4</sup> "Dtsch. Zeits. f. Nervenk.," Nov., 1906, Bd. 31.

<sup>5</sup> "Deutsch. Zeitschr.," Bd. 35, page 175.

<sup>6</sup> "Neurol. Centralbl.," April 1, 1904.

<sup>7</sup> Oppenheim, "Myasthenische Paralyse," Berlin, 1901.

involved show an extraordinary susceptibility to fatigue. Jolly's case showed regular muscular exhaustion under electrical stimulus, and this has been generally found in other cases.<sup>1</sup> In most instances there is a tendency to improve and to relapse, as in the case of Collins, in which case the special senses of sight and hearing also showed rapid exhaustion. E. F. Buzzard,<sup>2</sup> in a study of five cases, noted decided sensory disturbances of a tabetic distribution in one, and fleeting areas of paresthesia and analgesia in others. Localized atrophies are also possible and certain changes in the muscle fibers indicate early amyotrophic conditions. In all his cases various muscles and organs showed lymphocytic infiltration, though thymus glandular anomalies were not always present. Mental disturbance of a mild order has also been encountered.



FIG. 55.—Ophthalmic cycle in an advanced case. 1. Normal-like, extrinsic-muscle flex, drooping eyelids, etc.; 2. Deviated straight to extreme rigidity; 3. Forced straight to thoracic flexion and to gradual degree rigidity; 4. Forced the eyes with difficulty, the eyeballs were hardly covered.

The condition is marked essentially by nothemia, affecting particularly the motor apparatus. The prognosis is grave; a fair proportion of the cases terminate fatally through asphyxia. In the treatment, rest is advised with correction of any toxic or septic factor that may be discovered. The free administration of strychnin is probably harmful. I have seen the tendon reflexes repeatedly disappear under its use by the hypodermatic method, returning regularly upon its withdrawal. Spermin, electricity, massage, and prolonged rest were advocated by Wier Mitchell. (Personal communication.)

<sup>1</sup> Buzzard, "British Med. Jour.," March 3, 1900.

<sup>2</sup> "Brain," Winter, 1905.



## PART III.

# DISEASES OF THE BRAIN PROPER.

### CHAPTER I.

#### THE CEREBRAL CORTEX—LOCALIZATION.

**General Considerations.**—The subject of localization of function in the cerebral cortex has attained great importance and its literature vast proportions. The practical considerations will be briefly and somewhat dogmatically set forth. Many points are still under debate, awaiting further experience and experiment, and some of these problems it is likely will never reach solution. It is well determined that there is a definite area of the cortex that is closely associated with motor functions. As a working scheme we may consider that motion is represented in three levels: First, in the gray matter of the spinal cord; second, in the Rolandic area of the cortex; third, in the highest level of conscious thought, probably in the frontal region of the brain. The spinal level may be considered that of reflex, vegetative automatism, the Rolandic level that of motor combinations, and the frontal area that of conscious, selective, and intelligent action. Thus, destruction of the highest level leaves automatic action practically unimpaired, as in the experiments of Goltz, who removed the entire cerebrum of dogs without depriving them of muscular motion or bodily function. In the automatism of dementia the motor combinations are likewise preserved. The mid-level, the Rolandic region, may be destroyed, leaving consciousness of volitional motions and the will to execute them, but the cortical mechanism of their muscular production is gone, and they default, as, for instance, in motor aphasia. If the lowest or spinal level be destroyed, the mind and the memory organs have lost their tool and peripheral paralysis obtains. All thought contains the two ideas of motion and sensation. They cannot be separated, and without them consciousness is impossible. Indeed, they are in a certain sense identical. Motion is to the mind but the sensation of a change of position, and sensation is only the recognition of variations of motion. The flutist, by laborious conscious effort, establishes motor faculties in his central gyri which can subse-

quently be called into operation by the will with a rapidity of which conscious thought is incapable. The new-born child can hardly direct its hand to its face, but very rapidly develops a coordinate motor control of this act in the motor cortex that thereafter is easily recalled or subconsciously repeated. In the spinal levels single muscles or groups of muscles are represented. In the motor cortex coordinate and functionally associated movements are located, and in the highest level resides their volitional control and the power to recall and select them.

If these propositions are true in any degree, we would expect a neighboring relation of motion and sensation in the cortical representation, and this is no doubt the case. It is more than probable that sensation is represented bilaterally in the cerebral cortex, more completely than unilateral motion, and is consequently less modified by one-sided cerebral disease.

The inharmonious views variously entertained relative to sensory depots in the cortex may be partially reconciled if we consider the sensorimotor area as a midway station for sensation as well as motion and receive of a higher cortical sensory level.

The motor cortex is anatomically, or rather histologically, divided into from three to eight layers by various investigators. The important fact is brought out by all that the superficial layers are granular, and that the cells become progressively larger and more completely differentiated as we descend, until, in the lower strata of the motor region, the cells correspond in appearance to the multipolar pyramidal elements of the motor horns in the spinal cord. The cortical cells are practically all present at birth, and the development and growth of the brain depend mainly upon the increase in their dendritic processes and the fibrous feltwork that supports them. It is now generally believed that the interrelation of these cells is due not to actual continuity of their processes, but merely to their interlacing and apposition. The conditions, apparently, which best favor the transmission of nervous influence and the functioning of nerve-cells would be close filamentous apposition. Withdrawal of contact might serve as an insulating and inhibiting measure, a theory strongly supported in some quarters, but as yet only a theory. The nuclei of these cells are now considered as only dominating their nutrition, and not otherwise essential to their activity, which is relatively the same at the dendritic periphery as in the cell-body.

Movements dependent upon paired muscles, such as those of the trunk, are rarely abolished by unilateral brain disease. Those of a specialized and one-sided character, however, may be completely inhibited by unilateral disease of their cortical centers. Thus, the frontalis is rarely affected in cortical hemiplegia, while the unilaterally acting muscles of the lower part of the face are usually paretic in this condition. It is an acceptable proposition that all skeletal muscular activity is bilaterally represented, and it is also true that all unpaired muscles and their coordinate activities are more particularly controlled by the opposite half-brain. The acquired faculty of speech, however, and the numerous motor and sensory functions associated with it, are almost always



mainly represented in the left cortex in right-handed individuals. The superior weight and development of the left half-brain is probably largely attributable to its better nutritive supply through the arrangement of the vessels at the aortic arch and the larger caliber of the left carotid. This induces right-handedness, which in time, no doubt, retroactively increases the functional activity of the left cortex. At the same time the left hemisphere becomes potentially greater, more acquisitive, and therefore largely the seat of acquired motor and sensory education, which in turn increases its growth. It is probable that some overflow occurs



Fig. 56.—Scheme to represent the vertical arterial distribution. A.P.M., delivery of the *anterior posterior middle*; A.C., short arteries to the gray matter only; A.L., long arteries penetrating the white substance (after Broca).

in most brains, so that automatic emotional and explosive expressions either come to be located in the right brain or their frequent repetition sufficiently educates the right cortex to enable them to be recalled through its agency when the left centers are cut off. Similarly, if speech control be lost to the left half-brain, the right, especially in young persons, may be in turn educated to take its place in large measure. It is worthy of consideration whether the whole conscious and unconscious tendency of education, habit, custom, and practice to make all men right-handed is not a serious mistake. It seems plausible that should left-handedness, or rather ambidexterity, be assiduously cultivated in children, the two hemispheres of the brain might enjoy a greater equality and the individual secure not only amplified muscular control, but a certain lessened liability to aphasia and hemiplegic losses.

**Motor Cortical Localization.**—The human cerebral motor cortex has been mapped out with fair uniformity by numerous investigators. Following the lines laid down by Ferrier, Schaefer, and Horsley, originally based upon experiments on the monkey, the results of focal lesions in man and actual stimulation of the human cortex by electricity have given a fair degree of precision to the outline of the motor area. Comparing figures 67 and 68, we may see that all skeletal-muscle groups

are represented. Such outlines must be taken as suggestive rather than actual. There is no sharp boundary between the adjoining centers, and these fields overlap. The dipplings of the sulci also serve to interfere with sharp limitations of the cortical areas and obstruct the experimental stimulation of individual movements. Every muscular movement, apparently, has a focus of principal or major representation in the cortex, but such a movement is so wrapped up with other coordinate movements, and so widely related functionally, that its representation in a minor degree may spread over great areas. The thumb, for instance, is principally represented in a given small cortical center, but the prehensile action of the thumb is related to the grasp of the fingers, the fixation of the wrist, the rigidity of the whole upper extremity, and even to action of the trunk and lower limbs in strongest efforts, during which the opposite members also come into play.

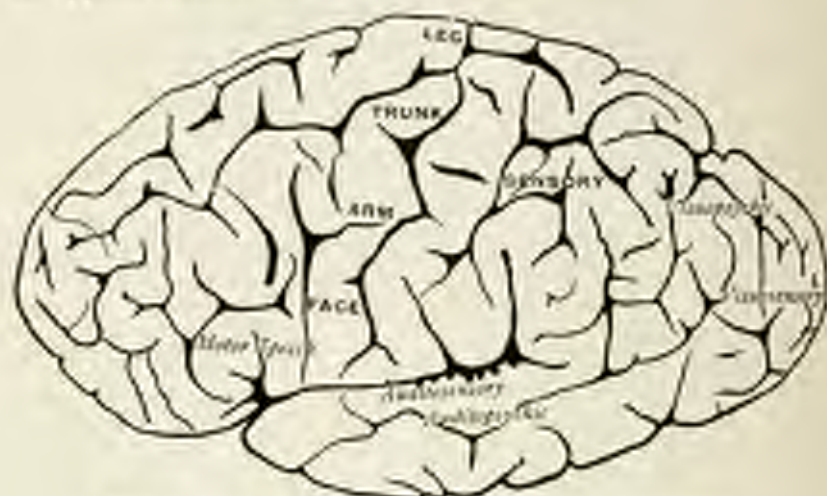


Fig. 42.—Functional areas of the cerebral cortex of the left side (after Campbell).

The most recent investigation of cortical localization in anthropoids by Sherrington and Grünbaum, and the histological studies of A. W. Campbell,<sup>1</sup> indicate that the true motor region of the cortex is much less extensive than was formerly thought. The functional groups of skeletal muscles are represented in the precentral or ascending frontal convolution from the lower end of Rolando's fissure up to the midline of the brain, and to a slight extent on the medial surface of the hemisphere, in a continuous narrow zone. The bottom of the fissure of Rolando sharply bounds the motor area behind, and it extends forward not to exceed the width of the precentral gyrus.

Liepmann<sup>2</sup> and Wilson<sup>3</sup> have shown conclusively that the ability to perform skilled movements with the limbs resides in the first and second

<sup>1</sup> "Histological Studies of the Localization of Cerebral Functions," 1906.

<sup>2</sup> "Montesani, C. *Psychiatr. u. Neurol.*," 1903, 1906, 1907.

<sup>3</sup> "Brain," 1908.



frontal convolutions of the left side. Their destruction results in a loss of such motions, a condition called *apraxia*, analogous to or identical with motor aphasia and agraphia. This state may be unattended by any evidence of paralysis.



Fig. 48.—Frontal view of the motor areas of the left hemisphere (after Langdon).

At the branching of the fissure of Sylvius motions of the *tongue* and *pharynx* are represented, and immediately above them the muscles of the *face*—first the lower, then the upper, face parts. Next in order we



Fig. 49.—Extension of body to vertical axis.

encounter *finger*, *wrist*, *elbow*, and *shoulder* movements; then those of the *trunk*, and highest of all those of the *lower extremity*, which lie over the medial margin of the hemisphere.

In front of the centers for the tongue and mouth parts, in Broca's convolution, the third left frontal, *motor vocal speech*, is principally represented. In left-handed individuals this motor function is located on the right side of the brain.

If we take the Rolandic area, from the superior margin of the half-brain to the Sylvian fissure, and divide it into fifths by horizontal parallel lines, the face occupies the lower two-fifths, the upper extremity the next two-fifths, and the trunk and lower extremity the highest and remaining fifth.

**Sensory Cortical Localization.**—The impossibility of accurately determining sensory disturbances in animals is one of the major reasons for the confusion that exists regarding the cortical representation of this function. Now that the unipolar method of stimulation is being adapted and the comparative insensitiveness of the brain has been proven we may expect definite records on many points of cortical function through observation on the human cortex of conscious subjects. The postcentral convolution, according to Campbell, and a large portion of the parietal area, possess the histology of sensory cortical structures. There is a large mass of clinical observations to indicate that this portion of the cortex has sensory function, though Bergmark<sup>4</sup> is disposed to limit it solely to the post-central convolution. *Stereognosis*, a complicated sensory function, is located in this region, and backward and below in the region of the angular gyrus of the parietal lobe on the left side we confidently locate the cortical representation of *visual functions*.

**Vision.**—The optic radiations arising from the outer geniculate body and in the neighborhood of the quadrigemina stream backward into the occipital lobe and reach the cortex at its apex. It may now be accepted that half-vision is represented at this location,—namely,—that the corresponding half of each retina is subserved by the occipital cortex of the same side (see Fig. 29). The investigations of Henschen and others indicate that the macula is represented in each occipital cortex in more or less complete degree. It thus results that ablation or destruction of one occipital lobe produces double homonymous hemianopsia. In this condition the macula is regularly spared, as it is sufficiently supplied by the opposite lobe. It is likely that the half-retina may be further divided into irregular and varying upper and lower quadrants, with localized cortical representation. It is indicated by some clinical cases that the fibers in the optic radiation which serve the upper quadrants are above those for the lower, and that the macular fibers are placed between them. Altogether they make a bundle about a centimeter thick, passing horizontally backward at the level of the second temporo-occipital gyrus. They finally reach the apical occipital cortex, and seem to have their maximum field in the neighborhood of the calcarine fissure on the nasal surface of the lobe. According to Holmes and Lister the macula is represented at the occipital apex and the upper quadrants of the half field are placed above the lower in the cortical field as in the retina and the optic radiations.

Higher visual coordinating and combining centers no doubt exist, and probably are in the angular gyrus of the parietal lobe, where Ferrier

<sup>4</sup>"Brain," 1910.



first placed vision. Functions related to printed speech are probably located here in particular and on the left side alone. Destruction of the left angular gyre, therefore, produces *word-blindness*, and destruction of both angular gyri produces *word-blindness*, all objects failing recognition.

**Hearing** is subserved by the first and slightly by the second temporal convolutions, which are in relation with both ears. It, therefore, requires bilateral destruction of the gyri to produce complete *cerebral deafness*. Functions related to the reception of spoken words are apparently represented in the posterior two-thirds of the first and second temporal gyri on the left side. When this area is destroyed, the right-handed patient becomes *word-deaf*.

**Smell and taste** are presumably located in the cortex of the median surface of the temporal lobe, smell in the uncinate convolution, and taste below it in the fourth temporal. Broca, and after him Zuckerkandl, located smell throughout the limbic lobe, but Ferrier and later investigators are disposed to confine it to the uncinate gyre and the hippocampal region. The recorded cases bearing upon the locations of smell and taste are extremely few and not convincing. The region is not often affected by limited lesions. A few hemiplegics lose smell in the nostril opposite a lesion which involves the temporal lobe. Lesions of the tip of the temporal lobe have also been found in epileptics who had a gustatory aura or one of smell. Insane hallucinations of smell have been related to disease in this portion of the brain.

**Cortex of Unknown Function.**—Examination of the diagrams (Figs. 67, 68) will at once indicate that the cortex of unknown function is much greater on the right than on the left side, owing to the fact that speech finds its representation almost solely in the left brain. In the frontal area, anterior to the motor zone, it is customary to locate the higher psychological functions. While it is true that this region may be largely destroyed by injury or disease without producing localizing symptoms, there is a rapidly growing number of cases indicating that mental and moral obliquities are usually the sequence of such lesions. Ablation of the prefrontal region in dogs and monkeys induces a change of character, of disposition, of behavior, that is clearly recognizable. In men with prefrontal brain injury, mental sluggishness, want of attention, diminished memory, loss of energy and of self-control, are noted with more than coincidental frequency. The upper and posterior parietal regions, a portion of the temporal cortex, and the island of Reil are still unaccounted for. These are known as regions of *latent lesions*—of lesions which do not necessarily produce symptoms.

**Cranio-cerebral Topography.**—It is very necessary in many cases of brain disease to locate the underlying cortex, either for the purpose of operation or to determine the relation of scalp wounds, depressed fractures, and other traumata, to the cortex. No brain presents symmetrical hemispheres, consequently we can not expect a close resemblance between the brains of different individuals. There are also variations for sex, age, and body-weight. Very often there are supernumerary or unusual convolutions on one or both sides. A number of methods have been devised by Reid, Horsley, Hare, and others, to map out on the scalp the underlying cortical areas. They all have valuable





*frontal lines* are drawn to the vertical point already mentioned. At the junction of the middle and lower thirds of the frontal line lies the fissure of Sylvius, and this point, corresponding fairly well to the squamous suture of the temporal bone, is called the *squamosal point*. From the squamosal point a line, the *oblique line*, is drawn downward and forward to the external angular process of the frontal bone, at the upper orbital border level, as in fixing the glabella point. Divide this line into twelfths. Five-twelfths of the distance from the angular point to the squamosal point begins the fissure of Sylvius; at seven-twelfths it bifurcates and extends by its horizontal line backward under the oblique line, and in its continuation to a distance posterior to the squamosal point equal to the distance of the point of bifurcation anterior to this intersection of oblique and frontal lines. At the termination of the oblique line the Sylvian fissure turns upward about  $\frac{1}{2}$  of an inch parallel to the frontal line, to terminate approximately under the parietal boss. To mark the fissure of Rolando, draw a line from the sagittal line  $\frac{1}{2}$  of an inch posterior to the vertical point downward and forward, at an angle of about sixty-seven degrees, to terminate  $\frac{1}{2}$  of an inch anterior to the squamosal point on the oblique line. The fissure of Rolando lies under this line, but terminates, as a rule,  $\frac{1}{2}$  of an inch above the Sylvian, though very rarely opening into it.

Having thus fixed these two important and tolerably uniform fissures, the marking off of the principal convolutions is simple. Owing to the fact that there is no absolute relation between the cranium and the convolutions, it is necessary, when they are exposed, to verify them, if they are motor in character, by the application of the faradic current. This is done by means of a wire pointed electrode, and may be accomplished even through the unopened dura. The exact localization of the gyr before the skull is opened is less important when a large motor flap is made, as most commonly is done.

## CHAPTER II.

## SPEECH AND THE CORTEX—APHASIA.

**General Conditions.**—A word has four principal cerebral qualities: it can be heard and seen, and it can be spoken and written. The first two are afferent qualities of perception. The second two are efferent qualities of expression. We, therefore, have four groups of word functions: (1) Those of sound, or *auditory*; (2) those of sight, or *visual*; (3) those of the motions necessary to express words in speech, *vocal motor*, and (4) probably those of the motions required to express them by written symbols, *graphic motor*. For these four groups we have four cortical areas, as indicated in Fig. 71, p. 174, where these word characteristics are principally represented. The speech area of the brain is an extensive one, and the faculty of speech in its various qualities is subserved by it in a general or common manner. The elements of motor or expressive speech, that is, vocal utterance, writing, and motor signs and gestures, pertain more to the anterior section of the speech zone, namely, in front of the fissure of Rolando. The elements of sensory or receptive speech are mainly represented in the posterior portion



Fig. 71.—Speech areas in the cortex. A, Auditory; B, motor vocal speech; C, visual speech, including motor characters and appositions. D, graphic motor speech (after H. Y. E).

of the speech area. These word-centers are brought into mutual relations by systems of connecting fibers and into relation with their corresponding receptive and emissive peripheral organs by afferent and efferent tracts. We may indicate these connections by a diagram (Fig. 72). Thus, take the word apple: when it is spoken we hear it through the temporal lobe; and when written, we see it through the angular gyrus of the parietal lobe. We call up combinations of muscular movements in the third left frontal gyrus when we would utter it vocally, and those probably in the neighborhood of the hand-center when we would express it by writing. At the present time the arbitrary and even partially theoretic division of the cortex into areas and depots for the



various verbal functions has undergone much modification as a result of the critical review begun by Marie,<sup>1</sup> who, indeed, denies all speech qualities to the third frontal or Broca's convolution. It seems probably true that all the cortex and subcortex associated with speech may be affected by a lesion in any part of it. Such lesions produce a general reduction of speech power which takes on various phases, depending to some extent on the location and extent of the lesion or lesions, and numerous variations are determined by the personal characteristics of the patient and the lapse of time.

Schematically, we may say that as one or another of these major speech-centers is diseased we have corresponding varieties of cortical speech defect, or aphasia. They are: (1) Auditory aphasia, or word-deafness; (2) visual aphasia, or word-blindness; (3) motor aphasia, or aphemia, and (4) graphic motor aphasia, or agraphia. Disturbances of the connecting fibers also disturb speech, giving rise to a number of *receiving* or *connecting* aphasias. Again, more than one center may be simultaneously affected, causing *combined* aphasias. The four primary

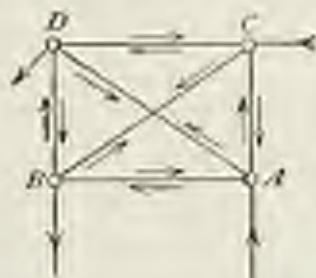


Fig. 26. Diagram showing receptive and expressive speech-motor, and the primary interconnections of the major cortical representations for speech. A, P.C., B, corresponds to the cortical parts similarly indicated in the preceding figure (after W. Miller).

so-called word-centers are, to a large degree, mutually dependent. Word-blindness is often attended by agraphia, as the mind is unable to recall the visual image of the word, which is to be copied, as it were, through the action of the graphic center. Again, if there is word-deafness, the patient cannot write to dictation, though he may copy perfectly.

Individuals vary greatly in the quality of their verbal faculties. As first emphasized by Charcot, one may have his words principally associated with the auditory area (auditive), another with the motor area (motor), and a third with the visual area. If we learn a new language by ear its memories are mainly auditory, and if by the look, they are visual. It is only when we have acquired oral proficiency that our motor faculties are fully established and may finally, perhaps, predominate. Probably only those who constantly express themselves in writing for many years so fully establish the graphic motor functions that they become relatively independent of the other word-centers. It follows that children always have auditory word memories first developed, the motor or verbal expressions following. The visual and graphic memories

<sup>1</sup> "Sem. med.," 1906, 1907.

then in men are acquired. From these considerations we can understand how it is that apparently identical lesions may produce results differing in degree in different cases.

The *stability of word faculties* depends upon the intensity with which they are imprinted upon the convolutions, either by some special emphasis or by frequent use and repetition. Thus, words as well as incidents that come with the shock, for instance, of fright, are always readily recalled. Every one knows the value of repetition in fixing a point in the student's mind. Nouns, being least frequently used, are the most easily displaced, and proper nouns suffer the earliest of all. Verbs, adjectives, adverbs, and prepositions follow in a methodical order, governed by the rule of depth of imprint due to repetition in daily use. This necessarily varies with the individual. An oft-repeated oath, "yes," and "no" are the most persistent of all.

It sometimes happens that the word or phrase on the patient's lips, at the moment of the stroke producing the aphasia, remains his only vocal expression. This he repeats at every attempt to speak, being, as it were, saturated or intoxicated with it. It has been suggested that its imprint under the circumstances of the stroke has made it indelible. In writing, the aphasic condition is often indicated by the repetition of letters or words. The name is usually signed promptly, if the patient has been accustomed to the act, which tends in time to become automatic. Sometimes only the first few letters of a word are accomplished, and then repeated again and again, or words are repeated. The tendency to *echolalia* is also very noticeable, the patient repeating some word or expression he has himself uttered or just heard others employ.

In many cases of aphasia the patient miscalls objects or uses wrong words, especially names and personal pronouns. This is denominated *paraphasia*, and is most marked in lesions of the auditory word memory depot. In this case the patient does not make persistent attempts to correct himself, as he is unable to recognize his own mistakes. When the auditory centers are not primarily involved paraphasia is recognized by the patient, who constantly manifests his annoyance at being unable to utter the proper word.

The *mental capacity* of aphasics varies greatly, and must be carefully estimated in a given case. As a rule, it is somewhat modified, as is clearly indicated by the changed temperament and disposition practically always present, even in the slightest cases. The organic mischief producing the cortical lesion may involve complete dementia, and in any event the mind works somewhat clumsily. According to Bastian and others the sensory forms of aphasia, those mainly marked by auditory and visual disturbance, are more likely to present mental deterioration and to develop insanity than the motor varieties. In such cases hallucinations of sight and hearing, due to irritation of the cortex, are not infrequent. Even in cases of slight degree, as Marie has well insisted, there is a certain level of complexity of mental operations beyond which the patient cannot go. As ideation depends so intimately on word processes, the reduction of the cerebral speech faculties in any manner necessarily impairs the flow of thought.

Besides the loss of visual word processes, the patient is unlikely to



recall figures, algebraic or other conventional signs, and musical notation. *Mind-blindness*, in which all objects are unrecognized, is due to a bilateral lesion of the visual cortex. In some cases of auditory aphasia the patient has shown a loss of musical appreciation, a condition termed *amusia*, and, as far as music is an acquired educated faculty, it is presumably associated with the left temporal lobe. Most motor aphasics whistle or hum tunes with more or less precision, and cases are on record in which motor speech was lost, but the patient could sing the words of a song correctly. One case replied "God damn" to every question, but got out most of the words of "Annie Rooney" with distinctness when he attempted to sing. Such cases demonstrate conclusively that word memories are not destroyed, but there is a general reduction of the speech faculty which, assisted by the crutches of rhythm and music, still functions.

Secondary means of expression, such as pantomime, facial expression, and gestures, are usually retained, but in rare cases the patient neither correctly produces nor understands them. This may go to the extent of nodding the head when negation is intended. The loss of gesture and mimicry is called *mutism*; their misuse is *pervervism*. Both these are manifestations of *apertion*. Emotional facial expression is generally retained, so that when the mind is not too much impaired the feelings of the patient are vividly portrayed in his countenance. There is some reason to locate these expressional centers in the basal ganglia, especially in the optic thalamus.

The motor cortex subserving speech is all within the domain of the middle cerebral artery, and aphasia is most frequently due to diseases of the branches or the trunk of this vessel. Trauma, meningitic disease, and new growths may also cause it. The most common simple variety of aphasia is the motor form, next the auditory, and then the visual. Distinct cases of graphic motor aphasia are extremely rare, but a convincing instance has been recorded. A combination of motor aphasia with the auditory and visual varieties is that ordinarily encountered. In this connection the various speech areas may be affected in different degree. Variations due to improvement or failure in the various speech qualities in any given case furnish constant study. The initial conditions are usually exaggerated by the participation of surrounding brain areas in the field of cerebral insult. After a few weeks or months, through education of the opposite cortex and partial local recovery, the clinical picture may be entirely altered.

Before taking up the various schematic forms of aphasia the reader is referred to the methods of examination in such cases described in Part I, p. 69. It may be well to call attention to the fact that the blind read type with their fingers, and that the deaf, by lip reading, cultivate special verbal processes, which must be specially localized in the cortex. We must also bear in mind that the word and the object are not the same, but that one is the symbol, sign, or name of the other. The word-percept is, therefore, distinct from the object-percept, which is a part of the higher intelligence. It follows that the aphasic can mentally recall the object, as a chair, though he cannot name it. The

object-percept may also be called up by various routes besides those of hearing and seeing. By smell, or touch, or taste alone we can recognize familiar quantities that have corresponding qualities, and at once the word or name springs to the lips. The object-percept is, therefore, made up of and embraces all the attributes of the given object with which the individual is definitely acquainted.

**Auditory Aphasia.**—The lesion is in the left temporal lobe. The most striking condition is the *word-deafness*. The patient gets only a partial suggestion of what is spoken to him, or fails completely. When the intellect is fairly clear he is likely to make good guesses from the circumstances of the interview, the examiner's gestures, intonation, expression, or the motion of his lips. These sources of suggestion must be guarded in making tests. Usually, the visual speech-center, from its near location, suffers with the auditory, so that alexia is added, but if it escapes, the patient may read understandingly.

As most people are strongly *orality*, the destruction of their auditory word memories notably impairs the emission of vocalized words. This manifests itself in paraphasia, and the speech may be reduced to a jargon or gibberish, which the patient expresses in a well-modulated way, and with an intelligent appearance. He does not recognize his own mistakes because of the word-deafness. He is likely to coin words and to repeat syllabic sounds. Frequently he starts a word properly and then mutilates it. In trying to describe his feelings, such a patient said, with gestures to his head and abdomen, "I have a solitary pretty well dear swell maneuver, and there are fullis things that hang solilar. There is a clean, flesurable, pleasmable, fair, unsarizable, and any samild-way for a good deal insurations that is totality all the time." He was annoyed that others could not understand him, and that he could not fully understand others. Very rarely would he attempt to correct a word. The writing of such a patient shows the same thing as his vocal utterances, both indicating the loss of the auditory speech memories upon which the majority of mankind are mainly dependent. He, therefore, uses wrong words, repeats words or letters, or his attempts lose all semblance to written speech. The ability to repeat words from dictation, or to echo sounds, may be destroyed, and is always greatly impaired. Copying written or printed characters is not affected. Anæsia may be present. The mental reduction is always pronounced.

**Motor Aphasia, or Aphemia.**—The lesion is in the foot of the third left frontal convolution, but usually extends to the foot of the ascending frontal, giving rise at the same time to motor difficulties in the lips and tongue. A subcortical lesion has all the significance of one purely cortical, or even more so, as a purely cortical lesion of the third frontal, producing motor aphasia, is by some entirely denied. A single case by Dejerine can be adduced. The characteristic condition in marked cases at first is the inability to produce articulate speech. Remnants like yes and no, or expletives, or an occasional habitual word may be retained, represented, as it is supposed, through the overflow into the right half-brain which occurs in the process of cortical ablation. As the right side is further educated, or if partial recovery is



the left side eases, the vocabulary increases. In the young the education of the right convolutions goes on with considerable rapidity if the mental impairment is slight. In recovering and in partial cases attempts at articulate vocal expression partially fail, from the loss of the verbal motor combinations. In severe cases the condition is absolute, *complete aphasia*. Motor aphasics sometimes misuse nouns (*paraphasia*), and they at once recognize their errors. They frequently paraphrase to get around their naming defect, as, for instance, "Give me some of that stuff for the bread," meaning butter. Wylie also calls attention to the infantile characters of the speech in improving motor aphasia, such as liping, lilling, and the cutting off of terminals and initial syllables. They cannot repeat dictation any better than they can speak spontaneously. As a rule, *agraphia* is present and proportionate to the motor aphasia. For this a number of reasons are assigned.

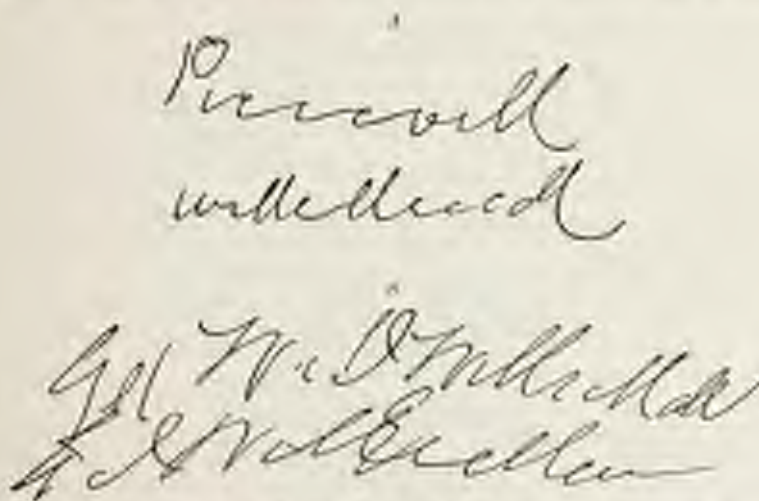


FIG. 74. Handwriting in a case of motor aphasia. Patient's attempt to write his own name—*A*. W. Whistler. *B*, after dictation.

Usually there is right hemiplegia, and the right hand is powerless. When the hand is not disordered, the agraphia persists none the less. It will be recalled that the graphic motor word memories are last acquired, the least deeply imprinted, and probably very easily disarranged. They are, no doubt, closely associated with the motor vocal memories, not only by nearness of location, but by function. As one learns to write, and even after much usage of the pen, he unconsciously inwardly pronounces first the letters singly and later the syllable sounds as he executes the written characters. Agraphia is really apraxia, and probably due to the participation of the precentral area of the second frontal gyrus. A few rare cases of motor aphasia have retained fairly good use of written speech. It may be that the individuals possessing deeply imprinted auditory, visual, and graphic-motor word memories would be less affected than the usual auditory-motor class. In attempts at writing,

motor aphasics frequently repeat a word or letter, or write only the few words of which they are vocally capable. Their names are usually signed with readiness if they can guide the hand. The motor aphasic who is able to use his right hand can copy readily and accurately, though he usually is unable to read. Even with the left hand they can copy as well as another. From dictation their efforts are no better than in spontaneous attempts. The motor aphasic understands all he hears and obeys commands unless too complex. There is no word-deafness.

Reading is notably impaired and in the same degree as writing. The association of visual speech with motor speech is a close one. All in learning to read pronounce as they go along, and even in after-life, when one is reading carefully, the tongue will be found slightly moving in the mouth in the same manner that the words would require were they pronounced. Yet many of these patients will intently scan the papers for hours or apparently read and reread the same books and insist that they understand what they read. They almost invariably recognize their own names, and in some cases seem to get the "drift" of what they read.

Motor aphasics showing a high degree of verbal difficulty are the ones in whom *anomia* and *paranomia* (*apexia*) are usually observed. Occasionally they cannot voluntarily protrude the tongue.

**Visual Aphasia.**—A lesion destroying the angular gyrus on the left side produces the peculiar defect of visual aphasia, or word-blindness.



Fig. 75.—A lesion (X) involves the optic radiation within the occipital lobe, producing hemianopia and word-blindness if acute (Sperner).

If this lesion extends deeply enough to involve the optic radiation streaming from the basal ganglia to the occipital cortex, hemianopia is added. A lesion in the optic radiation within the white matter of the occipital lobe may involve the connecting tracts between the half-vision centers in the apex and the higher visual centers in the angular gyrus, producing both hemianopia and word-blindness. It thus appears that word-blindness is due to disturbance of the angular gyrus alone, and that associated hemianopia is present only when the lesion implicates the optic radiation (see Fig. 75).

The visual aphasic can see perfectly anything put before him, but written symbols, figures, and other conventional signs have entirely lost



their significance. That he sees them clearly is shown by the fact that he can copy or draw them with as much precision as ever. Spontaneous writing, however, is lacking. Not being able to call up the visual images of written speech, he is unable to exteriorize them through the motor writing apparatus, excepting in the case of some automatic combinations, such as his signature. For similar reasons he cannot write from dictation. The hemispheric cases, without cortical or subcortical parietal lesions, write spontaneously and from dictation with ordinary facility, as, in these cases, the visual word memories are not necessarily destroyed, but only cut off from the lower half-vision cortex in the occipital apex. They are unable, however, to read what they have written. Spoken language is both understood and expressed with complete readiness. When familiar objects are not recognized, mind-blindness is present and the lesion is probably bilateral. It appears that ordinary visual sensations, as sensation in general, are symmetrically represented. The educated faculty of visual word memories is represented in the left angular gyrus alone.

**Graphic-motor Aphasia.**—Regarding this variety of aphasia there has been much dispute and uncertainty. According to some, Wernicke in particular, the act of writing is but the act of tracing the visual word memories. He points out that this can be done with the left hand, the elbow, the foot, or even with a pencil held between the teeth. Exner maintains that there is a graphic-motor center in the foot of the second frontal convolution adjoining the centers for the hand, but this is not sufficiently verified. The case reported by Goodinor,<sup>1</sup> however, is very convincing. In this instance the circumscribed lesion, a small new growth, was located in the foot of the left second frontal convolution. The facility with which the right is used as compared with the left hand, or the other portions of the body, indicates that it has attained a proficiency quite its own, which must be resident in or near its principal cortical representation. Some persons write with small finger movements, others with a wide, full arm-sweep, the pen being simply grasped with the hand, which is largely guided from the shoulder. An ambidexter uses either hand. Their cortical writing apparatus must vary correspondingly. In persons deprived of both hands, who write with the foot, quite another cortical area must be educated for the purpose. It seems probable that in every case this education would fall upon the corresponding motor cortex, which develops specially intimate relations with the area of visual speech. Even this relation is not absolutely required, as the blind accurately reproduce the letter forms they have learned by the sense of touch.

It seems unnecessary to presume the existence of a higher graphic-motor center. Agraphia, like motor aphasia, is a variety of apraxia, and is due in all probability to the involvement of the second frontal convolution. Agraphia is common in motor and rare in auditory aphasia. In auditory aphasia we more commonly have paragrammia, unless the lesion also involves the visual speech-cortex.

**Conduction Aphasias.**—The aphasias thus far considered are due to

<sup>1</sup> *Am. Jour. Med. Sci.*, May, 1899; *ibid.*, Sept., 1902.

lesions affecting more or less definite cortical functions. There are others due to lesions of the conducting tracts that bring these cortical areas of major functional speech representation into mutual and co-ordinate relations. The most important is the one produced by breaking the path between the auditory and motor word-centers.<sup>1</sup> This is usually due to a lesion of or in the neighborhood of the island of Reil. It was first described by Wernicke, and is sometimes called Wernicke's conduction aphasia. These patients present no particular auditory or articulative difficulty, but, owing to the loss of correlation between auditory and motor elements, they lose selective ability when they try to express themselves, and a most marked paraphasia and paragrammia result. They are obedient to direction, but cannot repeat dictation orally or in writing, though they copy with perfect precision. Neither can they read or pronounce aloud, though they seem to read understandingly. There is very little attempt to correct errors of spoken or written speech. The writer has seen such a case, due to traumatic hemorrhage, which was relieved by operation, the clot being found under the operculum, on the surface of the insular convolutions, where it had been located from the symptoms. The recovered patient now states that there was considerable mental confusion during the aphasia, to which the verbal disturbance no doubt would conduce.

Wernicke, Lichtheim, Wyllie, and others describe four other more or less theoretical varieties of conduction aphasia depending upon the location of the lesion in relation to the conducting tracts to and from the auditory and motor fields. Some quoted cases also are given in their support.

**Combined Aphasias.**—It has been pointed out that all word representation is in the arterial territory of the middle cerebral. Consequently from this source, as well as by trauma, meningitis, or cerebritis, they may all be thrown out at once. Simultaneous injury to the auditory and visual word functions is comparatively frequent, and it has been seen that graphic-motor activities usually disappear with the vocal motor—a combination due not only to association in function, but to proximity and to the attending apraxia. The loss of auditory and motor memories practically entails a loss of all speech attributes, as the visual and graphic-motor features are so thoroughly dependent upon them. Again, the centers may be unequally affected, so that sensory disturbance preponderates over motor, or the contrary. The type cases indicated in the foregoing pages are indeed rare, but by their description we can unravel the combined forms.

The mental disturbance is usually proportionate to the speech defect, and in total aphasia is very marked.

**Reeducation of Aphasics.**—One of the most important questions in a given case of aphasia regards recovery from the speech defect. Pure motor aphasia is perhaps the most hopeful variety in this respect, a verbal motor activities are the most easily built up. Reading usually follows much more slowly and writing is even more tardy. The forms of aphasia connected with the loss of sensory word attributes are the most persistent. As often mentioned, a majority of persons are solitary



DIAGNOSTIC TABLE OF BIVALENT ARTHRODIA

VENERY	Larval location	Parasit. Prev. type	Larval location		Larval location		Larval location	Larval location	Larval location	Larval location	Larval location	Larval location
			Epithel.	Metan.	Epithel.	Metan.						
Arctostaphylos	Posterior third of the third and second and third temporal gyr.		No.	Yes.	Parasitism — jagged.	Parasitism is at acrophis.	No.	Yes.	No.	Yes.	No.	Arctostaphylos, Metan. (not in mouth)
Arctostaphylos	Post of third and fourth gyr.	Parasitism.	Yes.	Parasitism.	Arctostaphylos (not in mouth).	Parasitism is at acrophis.	No.	Yes.	Yes.	May do so.	Arctostaphylos (not in mouth).	Arctostaphylos, Metan. (not in mouth).
Arctostaphylos	Angular gyrus of half posterior lobe.	Wood-McClure.	Yes.	No.	Yes.	No.	Yes.	Yes.	Yes.	Parasitism, but not in mouth.	Yes.	Arctostaphylos, Metan. (not in mouth).
Arctostaphylos	Island of Reil gyrus, anterior, just out of occipital lobe, tract between auditory and motor word centers.	Parasitism.	Yes.	Yes.	Parasitism.	Parasitism.	Parasitism.	Yes.	Yes.	Parasitism.	Yes.	Arctostaphylos, Metan. (not in mouth).

and all are necessarily so in childhood, though the child intently watches the lips of those teaching it to speak and probably acquires visual memories in association with the auditory impressions. In the sensory aphasias the mental disturbance is usually greatest. The loss of these earliest and usually most deeply grooved memories, which become dominant in the speech mechanism, is the most difficult to overcome.

The first step is to determine by which route the intelligence may best be reached. Even when both auditory and visual memories are gone, some patients can use the sense of touch to good advantage, and it will often be found an aid to put familiar objects into their hands when encouraging them to name them. If they are capable of giving attention, much may be expected. Simple sounds, such as a child first utters, like *ba*, *pa*, *ma*, may be indicated to them and repeated by the voice and in writing, by the position of the lips, mouth, and tongue of the instructor, or by taking the patient's hand and tracing the letters either in air or on a blackboard. If some object can be used,—say, a knife or pen,—it should be kept before the patient and placed in his hand when he attempts to name it. When simple sounds are mastered they can then be grouped into words, and the words associated with objects or actions, and so a vocabulary built up, which must be frequently and repeatedly and patiently rehearsed. By unremitting, intelligent effort, some enusive speech may be taught almost every case. It adds greatly to their comfort and makes their care less burdensome.

### CHAPTER III.

#### THE CEREBRAL WHITE MATTER, BASAL GANGLIA, AND CEREBELLUM.

DESCENDING from the cortex in converging lines we have the *corona radiata*, the fibers of which bring the brain-mass into relation with the lower brain parts and the spinal cord. Through the corpus callosum the homologous cortical elements on the two sides of the brain are brought into harmonious relation. The descending tracts reaching the basal ganglia are condensed into the *internal capsule*, in which the cortical motor fields are represented from head to foot, in an order from before backward, as indicated in figure 76.

The *internal capsule* lies between the lenticular part of the striate body externally and the caudate nucleus and the optic thalamus on its median aspect, but is not dependent upon them. Its anterior portion, or limb, is supposed to contain psychic paths to the frontal lobes. Lesions of this part of the *internal capsule* produce no distinctive symptoms. The motor routes lower down are continued in the lower portion of the crus and so on through the pons into the medulla and cord. The motor paths and their relations are indicated in figure 77, which shows that the face mechanism is inferiorly situated in the cortex, anteriorly in the capsule, and internally in the crus.

The sensory paths, situated in the posterior columns of the cord, pass upward into the posterior third of the hinder limb of the *internal capsule*. They reach the cortex of both hemispheres, according to Brissaud



and others, directly on the same side and indirectly on the opposite side, by sending off a branching path through the corpus callosum, as shown



Fig. 76.—Arrangement of motor paths in the cerebral cortex (after Sherrington).

in figure 78. Bilateral sensory representation is thus provided for. Doubtless motion is originally equally symmetrical in its cortical repre-

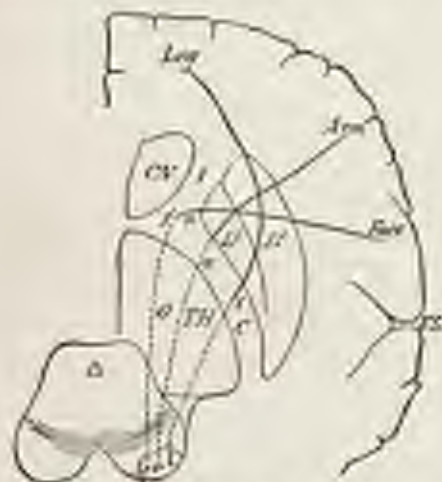


Fig. 77.—Diagram to show the relative position of the several motor tracts in their exit from the cortex to the brain. The section through the cerebellum is vertical; that through the internal capsule, I. C., horizontal; that through the crus is again vertical. CV, crus cerebelli; D, TH, optic thalamus; U and LA, the middle and outer parts of the lateral nucleus; C, a, l, arm, and leg fibers. The words in italics indicate the corresponding cortical centers (after Sherrington).

sentation, the apparent functional difference arising from the specializing of unilateral motility in the opposite or most intimately related cortex, by practice, habit, and education.)

From these diagrams we can understand that lesions in the cerebral white matter must be close to the cortex or in the internal capsule in

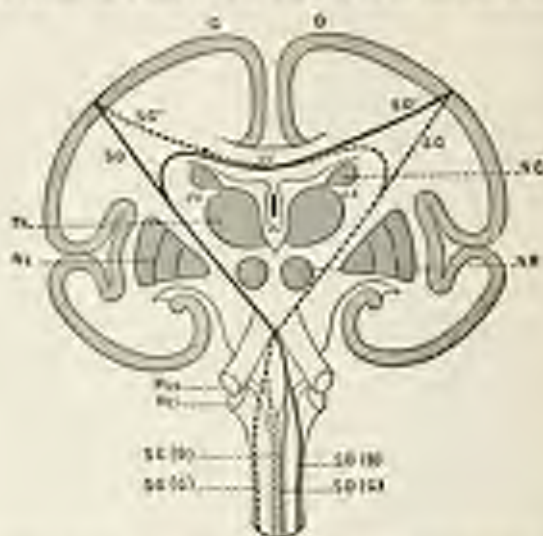


Fig. 15.—Schematic vertical transverse section of the hemispheres passing through the internal capsule and representing the sensory pathways. *Sc*, left hemisphere; *R*, right hemisphere; *C*, corpus callosum; *O*, internal capsule; *IC*, sensory pathway from left side of cord; *SC*, sensory pathway from right side of cord. Both sides are brought into intimate relation through the corpus callosum, and the sensory representation is bilaterally bilateral (after Brissaud).

produce permanent and definite sensory symptoms, as otherwise placed they do not intercept the pathways to both hemispheres. The symptoms in subcortical lesions correspond to the function of the related cortex, and in capsular lesions to the function of the particular tracts involved. The radiations to and from the cortex in the area of latent lesions may be, and often are, involved without giving rise to any symptoms.

The corpus callosum may be diseased to almost any extent without presenting any peculiar symptoms. Bristowe, Sharkey, and Schaffer<sup>1</sup> contend that tumors of the corpus callosum are likely to produce hebeticity, apathy, and prolonged placid coma. If its function is but to furnish correlating tracts between the two sides of the brain, its destruction would not materially interfere, perhaps, with the independent action of the half-brain. Putnam and Williams<sup>2</sup> note that in tumors of the corpus callosum mental changes are the only common symptoms, these consist in changes of character, impairment of memory and mentality, stupor, hallucinations, irritability and attacks of excitement. Epileptoid attacks, hemiplegia passing into diplegia most pronounced in the legs, and a peculiar ataxia or apraxia, a sort of lack of balance, and a poverty of movement have been variously recorded.

Lesions involving the optic radiations in the occipital lobes produce hemianopsia, and when on the left side they cut the radiations from the occipital apex to the angular gyre, word-blindness ensues, as described on page 180. Lesions beneath the auditory word-centers likewise produce word-deafness.

<sup>1</sup> *Rev. Speculant*,<sup>2</sup> Vol. XXV, p. 2. <sup>2</sup> *Jour. Nerv. and Ment. Dis.*, Dec., 1901.



The function of the *corpora striata* is still a matter of speculation. Lesions affecting them, however, almost invariably implicate the capsular tracts. In this locality we encounter the hemorrhagic lesion that is prominently causative of apoplectic hemiplegia. Whether or not posthemiplegic choreic movements and athetosis are due to disease of the basal ganglia cannot be definitely stated, but lesions in this locality are frequently attended by such motor disorders. S. A. K. Wilson<sup>2</sup> groups a number of cases under the title of *progressive lenticular degeneration*, a familial disease associated with cirrhosis of the liver. He defines this disease as one which occurs in the young, is often familial, but not congenital or hereditary; is essentially a disease of the extrapyramidal motor system characterized by involuntary movements, such as tremor, dysarthria, dysphagia, muscular weakness, spasticity, contractures, and emaciation. There may be emotionalism and mental symptoms. It is progressive and fatal. It presents bilateral degeneration of the lenticular nucleus and cirrhosis of the liver, of which latter feature no clinical symptoms obtain. The clinical picture is very definite and is commonly denominated Wilson's disease. The *corpora striata* are also probably diseased in Parkinson's disease or paralysis agitans (Hunt).

The *optic thalami*, when diseased, give rise to no definite symptoms if the lesions are confined to their anterior portions and do not invade the capsule. When the hinder portion of the optic thalamus is involved we are likely to have either a crossed blindness or a double hemianopia, probably from simultaneous injury to the optic tract. Bechterew claimed that facial emotional expressions are controlled by the thalamus in some manner. Brissaud, in cases of postparalytic spasmodic crying and laughing, locates the lesion near the knee of the internal capsule. The loss of facial emotional expression at least points to the internal capsule and the optic thalamus rather than to the cortex. Roussey, with whose conclusions Dana<sup>3</sup> is in substantial accord, outlines a somewhat definite thalamic syndrome. It is characterized—(1) by slight hemianesthesia and hemiparesis, without contractures and persistent exaggeration of the reflexes; (2) by hemitaxia, hemistereognosis, persistent pains, and paresthesia on the affected side, and (3) a tendency to choreoid and athetoid movements. To these Head and Holmes<sup>4</sup> add a tendency to react excessively to unpleasant stimuli on the affected side. The prick of a pin, painful pressure, excessive heat or cold, produce more distress than on the normal side of the body.

The *corpora quadrigemina* are seldom singled out by brain-lesions. When involved, adjoining structures almost invariably suffer, so that the resulting symptoms are difficult to analyze. The anterior pair are associated with vision, and apparently with some ocular movements. The pupillary reflex and movements of the eyeballs have been bilaterally impaired in some cases when they were diseased, and nystagmus, enfeebled vision, and blindness have been noted.<sup>5</sup> The posterior pair are thought to be related to hearing and equilibration. When they are diseased, the adjoining portion of the middle cerebellar lobe is usually involved, and probably gives rise to the symptoms mentioned. Raymond<sup>6</sup> contends that circumscribed lesions limited to the region of the

<sup>1</sup> "Brain," March, 1902. <sup>2</sup> "Ann. Amer. Med. Assoc.," Dec. 18, 1909. <sup>3</sup> "Brain," 1911.

<sup>4</sup> "Brain," "Rev. Spemann," Dec., 1909. <sup>5</sup> "Leprie," Paris, 1893.

corpora quadrigemina are prone to present (a) diminution of visual acuity, (b) palsies of associated ocular movements, (c) papillary anomalies, (d) paresthesias on one or both sides of the body, (e) ataxic disorders of movement in the limbs, (f) athetoid and choreiform movements, (g) contralateral diminution of hearing, (h) rarely disturbance of mastication by implication of the motor root of the fifth cranial nerve.

The *crura cerebri* contain the motor tracts on their under portions and the sensory tracts above. Crural lesions, therefore, produce hemiplegia of the face and limbs on the opposite side, marked by hemianesthesia if the sensory paths are at the same time involved. The proximity of the third nerve, as it issues from the inner border of the crus, lays it liable to damage at the same time, and then we also have an ophthalmoplegia. This affects the eye on the same side as the lesion and opposite to the paralyzed limbs—the Benedict-Weiser syndrome. When the lesion is deep, it gives rise to nuclear disturbance, as described under Diseases of the Cranial Nerves.

The *pons Varoli*, when diseased, often presents characteristic symptom groups that make the localizing diagnosis comparatively easy. It will be recalled that besides giving passage to the pyramidal tracts, which course downward from the crura to the medulla oblongata, it is traversed by the root-fibers of the fifth, sixth, and seventh pairs of cranial nerves from their nuclear to their apparent origins. The course of the facial fibers and their decussation in the substance of the pons opposite the apparent origin of the fifth pair are described on p. 121, and shown in figure 42. If a lesion falls upon the facial fibers before they decussate, and at the same time involve the pyramidal tract, which decussates lower down in the medulla, a paralysis of motion for the face and limbs on the side opposite the lesion ensues. If the lesion occurs below the facial crossing,—namely, in the lower third of the pons,—it will affect the face on the same side and the limbs on the opposite side, producing a crossed or alternating paralysis. Whether or not sensory symptoms are added depends upon the implication of the tegmental fibers, which lie above or behind the motor tracts. When this is the case the motor nucleus of the sixth nerve or its root-fibers is usually implicated at the same time, so that conjugate deviation of the eyes toward the side of the lesion and away from the paralyzed limbs is impossible. In destructive cerebral lesions higher up, it will be recalled, the ocular deviation is toward the lesion and away from the paralyzed limbs. Pontine disease may involve the motor speech-paths, which lie dorsal in the median portions of the pyramidal tracts, and give rise to articulative disturbance very like motor aphasia. Extension of the lesion dorsal and cephalad may involve the oculomotor centers.

The motor and sensory portions of the fifth cranial nerve-root may be involved separately or together, and trismus may be induced by irritation of the motor nuclei. A lesion which cuts the sensory root-fibers of the fifth induces anesthesia in the face on the same side and crossed paralysis in the limbs through injury to the pyramidal tract. Rigidity, spasm, and choreoid movements in the limbs are sometimes encountered, and convulsions, in acute disease, are common. If the middle cerebellar peduncle is affected, vertigo, vomiting, and tinnitus are usually present, and deafness on the same side may ensue. Irritative lesions of this



peduncle produce forced gyratory movements or forced one-sided positions in lying, which may be accompanied by corresponding positions of the head and eyes in the direction to which the turn is made. This may or may not correspond to the side of the lesion.

The *assilula oblongata*, owing to its small size and the vital importance of its nuclei, is most rarely invaded by acute disease without an immediately fatal termination. Disease of the olivary body may cut off the hypoglossal nerve and at the same time cause a crossed paralysis—the tongue on the same side and the limbs on the opposite. Diseases of this portion of the brain-stem are practically those of the lower cranial nerves and embrace the bulbar palsies already considered in Part II.

**The Cerebellum.**—According to Luciani, the cerebellum has for its main function the maintenance of sthenic tone in the muscular apparatus. If this be impaired, paresis, ataxia, incoordination, asthenia, tremors, and astasia result. It seemed probable that the cerebellum was practically of a uniform functional quality, which was quite evenly represented throughout its entire bulk, and that one part might take the place of another. Risien Russell and others have shown that the right lobe bears a certain relation to the right side of the body and the left lobe to the left side. Rothman contends that from before backward in the cerebellar cortex definite sections of the body are represented in the following order: eyes, head, face, jaws, tongue, neck, upper extremities and lower extremities, the trunk being represented in the posterior median regions. It is an experimental and clinical fact that cerebellar lesions of a sudden and extensive character at once produce very marked ataxic and paretic conditions, which may in time entirely pass away. Lesions similar or even greater in extent, but of slow development, may be entirely devoid of symptoms. It is evident that the cerebellum is capable of rearranging its functional relationships if gradually disturbed, and is of great recuperative powers after severe injury. Much confusion has arisen from confounding the symptoms of the secondary involvement of adjoining structures with those purely cerebellar.

We can now say that the right cerebellar hemisphere is in relation with the right side of the body and likewise with the left cerebrum. Mangazzini<sup>1</sup> has shown that injury to the thalamus induces atrophy of the opposite cerebellar half, and we thus have a crossed lesion, involving the cerebellum on one side and the cerebrum on the other. With the thalamic lesion the corpus striatum and motor cortex are usually involved.

A lesion in one lateral cerebellar hemisphere, if occurring with sufficient rapidity, as from hemorrhage or quickly developing abscess or tumor, produces *athetosis facii* on the same side of the body. This becomes manifest in one-sided muscular weakness or readiness of fatigue, in decreased coordination, in a tendency to stagger, and as the side of the lesion is the weaker side, the stagger is more marked in this direction,—that is, a patient with right-sided cerebellar disease is inclined to follow his right hand. Babinski,<sup>2</sup> under the name of *athetosciasis*, has particularized the difficulty presented by such patients in repeating a movement with rapidity and uniformity. This is commonly tested by ask-

<sup>1</sup>"Brit. Med. Jour.," May 18, 1895.

<sup>2</sup>"Neural Centralb.," Aug. 1, 1895.

<sup>3</sup>"Rev. Neurologique," Nov. 15, 1902.

ing for movements of pronation and supination of the forearm which quickly lose in uniformity and promptly subside from fatigue. A patient with cerebellar tumor found it impossible to use the salt and pepper shakers. The reflexes are also unilaterally reduced. At the same time the trunk may deviate to the sound side from the preponderating muscular tone on that side. Weakness of the ocular muscles on the same side as the lesion produces a tendency of the eyes to deviate in the opposite direction, and strong attempts to turn them toward the side of the lesion often develop nystagmic or jerky movements. It seems probable that lesions toward the head of the worm produce a tendency to fall forward, those toward the tail of the worm, a backward falling. These are the parietic manifestations. The cerebellar stagger and the ocular disturbances are often attended by vertigo of a pronounced subjective sort. Very commonly this is greatly intensified if the patient attempts to stand or even to sit up, and may prevent his doing so. In other cases, when the so-called cerebellar gait is well marked, there is no attending vertigo. Vertigo of a similar character may attend a tumor in the frontal region, which at the same time may cause an occipital headache and, according to Williamson,<sup>1</sup> in one-fifth of such cases induces bilateral weakness of the reflexes.

Goeden Holmes,<sup>2</sup> from a study of many gunshot injuries of the cerebellum, indicates that forced positions and movements are rare in man; that the position of the head is commonly one with the occiput drawn toward the shoulder of the homolateral or wounded side; that there is invariably atonia, asthenia, slowness of movement, and outward post-pointing on the affected side; that the eyes deviate away from the lesion and show quick nystagmoid movements when directed toward the affected side, with slow return excursions; that the tendency is to stagger and to fall to the affected side. His observations do not lend support to the localization of function in the cerebellum excepting the relation of each cerebellar half to the homolateral side, and he gives no particular importance to the vermis.

Irritative lesions produce another group of symptoms. They are marked by muscular stiffness in the extremities of the same side, by nystagmus, in which the jerk is toward the side of the lesion, and by such an arching of the body with the concavity to the diseased side as to occur on the affected side would produce. Eugyrosthenosis and episthotonos would perhaps point to the middle lobe or to both lobes. Drummond<sup>3</sup> has also noted convulsions of a tetanoid character on the same side as the lesion, and Ferrier has recorded them in animals subjected to operation. The activity of the lesion dominates the symptoms. They grade off in proportion as the diseased process is slow and may easily reach a vanishing point in chronic conditions that sometimes are astonishingly extensive.

A third group of symptoms arises from extension of the cerebellar disease to neighboring structures, or from pressure upon them. A one-sided cerebellar tumor, for instance, by extension forward above the medullary decussation, presses upon the motor tract from the vermis to the cord and gives rise to spastic symptoms on the side opposite the lesion, with increased myotatic irritability and even a tendency to con-

<sup>1</sup> "Glasgow Med Jour," Nov., 1899.

<sup>2</sup> "Brain," 1908, vol. xl, p. 48.

<sup>3</sup> "Lancet," July 28, 1904.



tractures. Pressure upon the floor of the fourth ventricle may affect the nuclei of the cranial nerves and give rise to paralysis of the fifth, sixth, seventh, eighth, ninth, tenth, and twelfth pairs of cranial nerves. The eighth or auditory nerve is particularly liable to be affected, and then aural symptoms are added. Tinnitus and vertigo of the Ménière variety may be superinduced, adding greatly to the complexity of the clinical picture. It is needful to investigate the aural condition very critically, as aural vertigo and cerebellar disease are often associated by the extension to the cerebellum of septic processes in the ear, and labyrinthine disease may closely imitate a cerebellar lesion. (See page 134.) Irritation in the fourth ventricle may produce polyuria and glycosuria. Obstruction of all Galenic veins produces dropsy of the ventricles, their distention, and all the manifestation of intracranial pressure. This condition is, however, more likely to be caused by occlusion of the iter from the third to the fourth ventricle by the same pressure which may interfere with the Galenic veins. Sudden death may follow disturbance of the pneumogastric nuclei. If the middle peduncle—the peduncle to the pons—be affected, forced versions result and forced positions are developed. These seem to be toward the opposite side if the lesion is irritative and toward the same side if destructive. Other clinical manifestations are those common to all intracranial diseases,—namely, headache, vomiting, optic neuritis, convulsions, and vertigo. Of these, an occipital headache is significant and is often associated with a rigidity of the neck and retraction of the head. Friedeberg<sup>1</sup> found this retraction marked in over half of the cases of cerebellar tumor in Aufrecht's clinic. Sensory disturbances are rare. Russell is inclined to think they may be present for a short time immediately after the onset of acute diseases, as hemorrhage and transient anesthesia are noticed in operated animals. Krüss,<sup>2</sup> from a study of ninety-seven cases of cerebellar disease, enumerates the frequency of symptoms in this order: "Headache, vomiting, optic neuritis, vertigo, ataxia, ataxia, ataxia, occipital pain and tenderness, inclination to turn toward the side of lesion, convulsions, and such secondary symptoms as nuclear paralysis, polyuria and glycosuria, tremors, and sudden death." Neither the mind nor the sexual desire is necessarily disturbed.

A combination of pontine, cranial nerve, and cerebellar symptoms is presented by tumors occurring in the cerebellopontine angle formed by the lateral lobe of the cerebellum and the medulla and pons. The fifth, sixth, seventh, and eighth nerves traverse this area and show various combinations of symptoms, depending upon their partial or complete implication. Encroachment of the growth upon the cerebellum adds symptoms of a corresponding nature and crossed palsies due to pontine disturbance are also encountered. Combinations of eye symptoms, such as squints and nystagmus, of ear symptoms, such as tinnitus and vertigo, of disturbance of sensation in the domain of the fifth nerve with diffuse symptoms of brain tumor and cerebellar indications, would furnish a syndrome almost characteristic of tumor in the cerebellopontine angle. The recognition of this syndrome is very important, as in tumor cases surgical technique and experience now offer a very favorable prospect to properly conducted operations.

<sup>1</sup>"Berlin. Klin. Wochensh.," Aug. 19, 1901.

<sup>2</sup>"N. Y. Med. Jour.," June 5, 1893.

## CHAPTER IV.

## FURTHER LOCALIZING CONSIDERATIONS.

Lesions of the brain may be broadly considered as *irritative* and *destructive*. From this point of view they respectively produce increased and decreased activity of function. We find the best exposition of these conditions in lesions of the motor cortex. Given a circumscribed definite lesion, such as that caused by a speculum of bone or a small tumor, that rather displaces than destroys the cortical elements in this region, and it is likely to so irritate them that increased activity is manifested in the peripheral area with which they are associated. A limited spasm or convulsion may ensue. If the irritation be too long maintained, necrotic or destructive cortical changes usually follow and are marked by diminished or completely lost peripheral function,—namely, paresis or paralysis. A lesion, at first irritative, may thus induce spasms in the hand, and after a time the hand becomes parietic if the progress of the disease reaches a destructive grade. Sudden destruction of the cortical mechanism, as by hemorrhage, causes immediate loss of power.

Were all lesions simply destructive or irritative, difficulty in deciphering them would be greatly reduced, but ordinarily they are combined in varying degree. Around a destructive process a zone of irritation brings new elements of disturbance into the symptom-field which, in turn, may be replaced by evidence of extending destruction. Again, in an area practically parietic from cortical disease, convulsions may occur, perhaps owing to irritation of remaining but inhibited cellular elements or from irritation of the subcortical tracts. In every case, therefore, it is highly important to know the clinical sequence of irritative and paralytic symptoms in order to determine the point of invasion, the progress of extension, and the limits of the lesion.

This brings us to the *invasion* or *extension* symptoms. These are transient in the widening convulsive manifestations of cortical epilepsy, and permanent in the slow encroachments of progressive disease. If we consider an irritant or discharging lesion to be located in a given part of the cortex, the disturbance to which it gives rise spreads in concentric and widening circles to the adjoining regions, which are successively upset, and the peripheral display is correspondingly and similarly broadened. The invading march of a Jacksonian fit can be foretold if we know its initial location or storm-center. Concentric rings on cortical diagrams enable us to grasp this point firmly. In figure 79, A, a fit starting in the arm-center would next call forth the face and head movements, then those of the trunk, and finally those of the lower extremity. Commencing in the lower extremity, the order would be reversed, as shown in figure 79, C. These sequences are in accordance with clinical facts. The order of convulsive invasion is not one of chance, but is rigidly dominated by the anatomical and functional rela-



tions of the cortex. The initial or signal symptom of a cortical fit, therefore, becomes highly significant as pointing to the storm-center, the point of greatest instability and usually the seat of organic disease.

Considerations of a similar character sometimes enable us, if we have all the clinical data available, to trace a neoplasm from its origin as it invades neighboring centers, and to relatively estimate its anatomical limits. The area of latent lesions must be kept in mind. It gives no special symptoms when traversed by the discharge of a fit or when invaded by a growth. The cortical fields of speech and of the special senses are subject to the same rules as the motor zone. Their invasion is attended by aphasic or sensory disturbances. In the latter case hallucinations are likely to appear, and may constitute the signal symptom of Jacksonian fits. Thus, patients may always hear a certain sound, see a certain spectrum, smell or taste a certain article as the fit comes on. In such cases the application of concentric cortical lines shows that the subsequent motor disturbance was subject to the same invasion rule that obtains when the storm arises in the central convolutions.

Peripheral sensory disturbance arising from cortical lesions is usually of a parasthetic sort. There may be some blunting of cutaneous, muscular, and joint sensations, but persistent anesthesia is extremely rare except in bilateral lesions. The double and complete representation of sensation has been sufficiently insisted upon (see p. 186). In Jacksonian fits the initial symptom is often a peculiar sensation or pain localized in or near the part that is first thrown into spasm. Patients are frequently at a loss to describe these sensations, and they vary from slight discomfort or slight formication to severe pain and intense burning. Their distribution is segmental or functional and does not conform to the peripheral nerve-supply. They are tolerably uniform in quality and distribution in any given case. Exhaustion of the motor apparatus after severe, and particularly after repeated, attacks of Jacksonian fits may lead to a paralysis lasting several days or weeks and a mistaken idea of brain destruction.



Fig. 72.—Jacksonian Epilepsy. To show the un-  
derminement of waves of discharge in the cortex,  
beginning respectively in the area, then, and  
by curves, 1, 2, 3, 4, 5, 6, and 7, by later  
discharges. (Figures 1 and 2 should be placed  
on the preceding page.)

From another point of view cerebral symptoms are *diffused* or *localised*. The great majority of endocranial lesions are marked by headache, vertigo, vomiting, mental disturbance, and often by optic neuritis. As these do not necessarily depend upon the part involved, they have little localizing value, but the presence of several of them is highly suggestive of brain disease.

*Headache* is a very usual symptom. It is pronounced in the cases which involve the meninges. Its importance in meningitis will be at once recalled. In syphilitic cerebral disease it has a marked tendency to come on toward night. Cerebral headaches are usually very intense and very refractory to sedatives. Lesions which do not impinge upon the cortex or meninges may give rise to no headaches whatever, unless pressure conditions arise, when the superficial portions of the encephalon are disturbed and pain ensues. Cerebellar disease is frequently attended by an occipital headache and a tendency to retraction of the head. The location of the headache is not always in close relation to the lesion. For instance, cerebellar disease has caused intense frontal headaches, and small tumours have given rise to a generalized head-pain. A circumscribed, deep-seated, persistent, and intense headache, however, has some localizing value.

Vertigo attends many brain disorders, and marks nearly all cerebral surprises arising either from without, as by contusion, or from within, as from embolism or hemorrhage. Implication of the nasal apparatus gives rise to the peculiar vertigo described under Ménière's Disease (see p. 134). A lesion of the middle cerebellar peduncle causes forced lateral movements, usually associated with vertiginous sensations, and cerebellar disease is marked by a stagger, into the causation of which some vertigo may enter. In a general way we may say that persistent vertigo is likely to be allied to diseases of the local parts. Those, too, may give rise to the ocular and labyrinthine forms from injury to the nerves entering the orbit or vestibule.

Vomiting in encephalic disease is a common symptom. The peculiarities which mark cerebral vomiting are lack of gastric disorder and nausea, a clean tongue, and the readiness and ease with which the stomach rejects its contents. There is very little retching, and the food or drink is regurgitated, sometimes with considerable force, in a *projectile* manner. Meningeal invasions of an inflammatory or other character often present this symptom. It is very common in disease of the corpora quadrigemina, the pons and the cerebellar regions, perhaps from more or less irritation of the pneumogastric nucleus.

All varieties of *mental disturbance* appear in organic brain disease. We encounter momentary unconsciousness in *petit mal*; *hététude*, and profound coma in meningitis, apoplexy, concussion, and intracranial pressure; maniacal disturbance after epileptic attacks; changed temperament and character after frontal lesions, and more or less dementia in nearly all cerebral paralytics. The diffuse cerebritis of general paresis presents its own usually highly colored mental picture. The mental attributes of aphasias have been already described. Mental symptoms have the same general significance as motor signs. Irritation means mental excitement, and overmental excitement leads to *hététude*, which



also directly follows pressure conditions, shock, and deranged cerebral circulation.

*Optic neuritis* in brain disease has been subverted to under *Diseases of the Optic Nerve* (see p. 100). Its presence in suspected intracranial tumor almost serves to clinch the diagnosis. The location of a growth or the position of an abscess, or their size, seems to have very little to do with the intensity of the papillitis, but it is most common in lesions of the basal ganglia and cerebellum. When unilateral, the growth is commonly on the same side of the brain. A neuroretinitis is a common symptom and sequence of meningitis.

The *localizing symptoms of brain disease* have been set forth in the three preceding chapters. In addition we often derive much information from the implication of the cranial nerves that takes place in their intracranial course. The various symptoms thus produced are discussed under *Diseases of the Cranial Nerves*. Diseases of the base, the peduncular, pontine, and medullary portions of the encephalon ordinarily have cranial-nerve concomitants. This is also true of lesions of the cerebellar peduncles and of other parts in the region of the series of cranial-nerve nuclei.

*Typical symptoms of some value* are often encountered in intracranial disease, and should always be sought. Trauma, if recent, are usually marked by bruises, wounds, or fractures that at once center attention on the underlying parts and on the opposite side of the brain where the force of the blow is expended. In later cases scars, cranial depressions, or evidence of bone disease are equally significant. The presence of *deformities* due to new growths and the conditions of the auditory canals, nasal passages, and pharyngeal vault are to be carefully noted. When the disease affects the meninges there is often *typical pain and tenderness* that can be elicited by making pressure over the scalp or by going over the surface with a percussion hammer. Its outline and permanence are suggestive of the extent of disease beneath. A neuralgia of the fifth or occipital nerve has its own tender points and anatomical outlines to distinguish it. By *percussion*, Macdonen, Starr, and others have been able to distinguish a different note over the seat of intracranial growths and diseases that were located close to the cranial wall. This, doubtless, requires a very acute ear, but should be sought in every suspicious case. Intracranial aneurysms may in some cases present a *bruit* that can be heard through the stethoscope. We would naturally expect the patient to be aware of it, as the conditions favorable for bone-conduction would be present. Two of the writer's cases of extensive intracranial aneurysm, in which a bruit was probably present, gave expression to no such subjective complaint, and auscultation was not attempted before operation. In a third, persistent throbbing had annoyed the patient for months and then disappeared, but the stethoscope revealed a decided bruit, of which she was not conscious.

In some cases of intracranial disease a *localized elevation of temperature* has assisted the diagnosis. Our present commonly available means of surface thermometry are deficient in accuracy. If the bulbs of clinical thermometers be passed through pieces of rubber protective and then

applied to the scalp by tight bandages, tolerably reliable readings may be obtained. It must be borne in mind that the left side of the head is usually, in the right handed, about one degree warmer than the right side, and that mental activity causes the temperature to go up nearly or quite another degree. Sometimes in suspected abscess a thermometer in each auditory canal may on one side show local heat even in the absence of any apparent local inflammation. Palpation may discover a brain tumor, as the cranial wall is often eroded by a cerebral growth, and that, too, when situated at a considerable depth. The *x-ray* has been used in many cases of brain disease. In a few instances it has aided in the diagnosis of tumor, but in cases of exostosis and other alterations of bones, in alterations of the sella turcica due to pituitary disease and tumors and in the case of foreign bodies, skiagrams are of the greatest value.

Finally, it is to be borne in mind that a brain-lesion located in a latent zone may give rise to symptoms at a distance. In some instances this results from interference with the blood-supply or the return circulation. In other cases the mechanism cannot be explained. In many cases where every symptom pointed to the cortex, the disease has been found deeply seated. The localizing diagnosis always contains uncertainties and should be expressed with a fair degree of reservation. Operations depending on it are, therefore, exploratory in every instance when external guiding signs are lacking.



## CHAPTER V.

## ARTERIAL BRAIN DISEASES.

In the preceding chapters of this part the localizing features of brain disease have been considered. They furnish the basis of the *localizing diagnosis*. An equally or more important question is that of the *pathological diagnosis*. What is the lesion? Comparatively few pathological processes are found in diseases of the brain, but their effects and results are numerous and serious. These diseases fall into groups related to: (1) The arterial supply; (2) the venous return; (3) inflammatory disturbances of the brain-substance, and (4) new formations. Surgical conditions, such as penetrating wounds, are left to weeks properly covering such accidents. We first turn our attention to the arterial supply of the brain.

**Anatomical Considerations.**—The arterial supply of the encephalon furnishes the anatomical basis of some brain-lesions, and presents practical points of great importance. The left carotid, leaving the arch of the aorta on a tangent that conforms to the natural blood-current, favors the passage of emboli of cardiac origin to the left brain. It will be recalled that all the blood to the brain proper reaches the encephalon by the internal carotids and the vertebrals. These four inlets are brought into intimate relation through the circle of Willis. From side to side compensation in the circle is practically complete in case the lateral arteries are occluded. From the carotid or anterior portion of the circle to the vertebral, basilar, or posterior portion, full compensation can not be provided owing to the smallness of the connecting arteries. From the circle of Willis two distinct systems of arteries arise,—the basal and the cortical. From the circle itself, and from about the first inch of the six great arteries, the anterior, middle, and posterior cerebrals, short, direct vessels plunge into the brain to nourish the basal parts, ganglia, and capsules. These anastomose but slightly with one another, and are of the nature of terminal arteries. Their occlusion or destruction irreparably cuts off the circula-

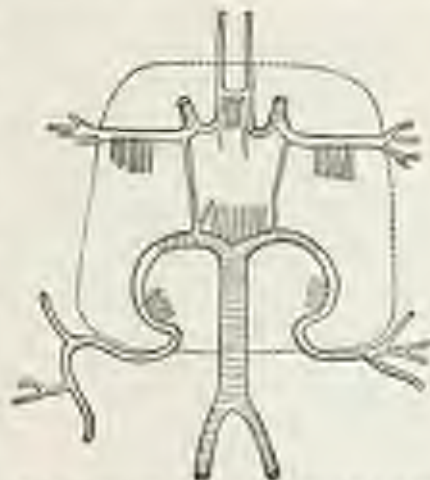


Fig. 96.—Osborne's diagram of the arterial base of the brain.

tion from a given portion of brain-tissue. Those distributed to the lenticulo-striate nucleus, the internal capsule, and a portion of the thalamus are derived from the stem of the middle cerebral. One, in particular, supplying the third layer of the lenticular ganglion, the capsule nucleus, and the upper portion of the capsule, is so commonly the seat of rupture that it was denominated by Charcot the artery of cerebral hemorrhage.



Fig. 81.—Distribution of the middle cerebral artery (Bass).

The main cerebral arteries coursing up over the hemispheres in the arachnoid have each a definite cortical territory, and these only slightly overlap. They give off two sets of branches,—namely, delicate arterioles, nourishing the cortex, which they enter at right angles, and larger straight, long branches, which pierce the cortex and supply the white matter of the cerebrum (see Fig. 86, p. 167). These pass inward and



Fig. 82.—Showing the distribution of the anterior, middle, and posterior cerebral arteries on the surface of the brain. The numerals I, II, III, IV indicate the areas supplied by the different branches; the dotted lines indicate the main trunks (modified from Meyer and Dehnbach).

almost come into anastomotic contact with the upward-reaching terminations of the capsular and ganglionic branches arising close to the circle. Between these arterial territories there remains an ill-nourished zone that is prone to senile softening (Fig. 81).

The cerebellum receives its blood-supply from the vertebrals and basilar; the medulla and pons are largely supplied by the basilar. It



will be recalled, from the description of the pial vessels (p. 75), that their cortical and deeper branches carry with them a periarterial lymph-sheath continuous with the pial space.

### CEREBRAL ANEMIA AND HYPEREMIA.

**Cerebral Anemia.**—Until recently an undue importance attached to the frequently alleged conditions of general brain anemia and hyperemia. They were spoken of as morbid entities, and diagnosis stopped at that. We must look upon them as purely symptomatic and secondary. In many respects they are marked by similar or even identical phenomena, and can only be distinguished by their associated states or incidental symptoms. The circulation of the brain, it will be recalled, is under the direct control of vaso-motor centers in the medulla and cervical cord.

It is exceedingly difficult, if not impossible, to say where physiologically increased blood-supply becomes a morbid congestion and entitled to the term hyperemia. Cerebral anemia is equally indefinite. In giving the causes of these opposite conditions, therefore, it has been found impossible to sharply separate the natural from the morbid states. Let us first consider the anemic brain.

**Etiology.**—Cerebral anemia is most common in infancy and old age, the periods of least brain activity. In old age it is the legitimate consequence of senile arterial degeneration. In adults it follows intense emotions, various peripheral, acute, painful disturbances, and shock of all sorts. Tobacco, ergot, belladonna, bromids, antimony, lead, chloroform, and many other drugs and poisons produce cerebral anemia. Fatty heart, aortic insufficiency, bradycardia, excessive hemorrhage, rapid evacuation of peritoneal and pleural effusions are attended by cerebral anemia. It takes a prominent place in all the general anemias, and in the pernicious varieties is especially marked and often attended by nutritional changes in the gray and white matter of the brain and spinal cord. Cachectic and exhausting diseases, such as cancer, typhoid, phthisis, etc., produce a similar condition. Ligature or obliteration of a carotid, or partial occlusion of both carotids or vertebrals by atheroma, serve to mechanically produce anemia of the brain.

**Symptoms.**—In acute forms of brain anemia, such as are typified by an ordinary fainting spell or a great loss of blood, the symptoms are



Fig. 81.—Schematic showing the anastomotic relations of the basilar and carotid arterial supplies. A, common location of senile infarcting; B, common location of cerebral hemorrhage (after Brissaud).

"darkness before the eyes," giddiness or vertigo, noises in the ears, feebleness, trembling of the limbs, nausea or vomiting, sometimes slight delirium, and then more or less loss of consciousness. Partial or generalized convulsive movements are common, and epileptiform convulsions are sometimes seen. Finally the condition remits or passes deeper into coma, and may terminate in death. The face is usually blanched, the pulse small and fluttering, arterial pressure greatly reduced, the pupils dilated, the skin covered with a cold perspiration. Similar conditions are seen in shock, and to a moderate degree in some cases of migraine.

In the *obscure form*, such as attends general anemia and the *cardexia*, patients complain of heaviness of the head, of headaches limited to a portion of the head, or a feeling of constriction about the head. They sleep lightly or brokenly and their sleep is disturbed by dreams. There is mental and physical inertia. They are irritable, peevish, impressionable, and scissilient. The pupils are dilated. Vertigo, sometimes ringing in the ears, and intense volitations are common, and often induced by rising from the recumbent position or by a quick turn of the head. In extreme cases there may be visual hallucinations and temporary blindness from retinal anemia. Slight delirium or maniacal states and even epileptiform convulsions are seen in severe instances. Optic papillitis or choked disc is occasionally encountered.

The diagnosis is not difficult except in the toxic cases. It must be remembered that the circulation in the face, or even in the retina, is not a reliable index of the brain state, but is sometimes a helpful guide. Anemia of the brain is relieved by placing the head low and intensified by the vertical attitude.

The treatment is that of the underlying causal state or toxic condition.

**Cerebral hyperemia**, like anemia of the brain, is always a secondary state, and, while arising from opposite conditions, has many similar subjective symptoms. It may be recalled that the passing from sleep to the waking state is normally marked by increased cerebral circulation, as is also the process of active mentation, which is attended by a slight increase in the surface temperature of the head, particularly on the left side in right-handed persons. The brain volume is augmented and intracranial cerebro-spinal fluid is displaced in cerebral congestion.

**Etiology.**—Muscular effort, coughing, vomiting, increased heart action, impeded respiration, a dependent position of the head,—all tend to increase the amount of blood within the skull and produce congestion or hyperemia of the brain. Usually the face shows a similar condition. Some families display a tendency to plethora and cerebral congestion. Menstrual periods are normally marked by an increased circulation in the brain. At the climacteric this occurs in flushes or waves and becomes very annoying. Gouty subjects are particularly liable to vascular disturbances, which frequently involves the cerebral circulation. Contracted kidney and arteriosclerosis are also commonly marked by such vascular storms. Insolation produces intense cerebral congestion, and exposure to cold has a similar effect by driving the blood from surface to center. In the same way during a chill the central organs are



hyperæmic. The nitrites, opium, alcohol, and the infections of typhoid, pneumonia, tetanus, and many fevers produce cerebral congestion. This may alone be the cause of the convulsions which so frequently mark the onset of eruptive fevers in children. The suppression of menstrual discharges or of chronic hæmorrhoidal bleeding and pressure on the vena by a tumor or focal accumulation raises the cerebral blood-pressure. Cardiac conditions may cause cerebral congestion by impeding the venous return or increasing the arterial tension. Inflammations about the head and neck and in the throat are commonly attended by cerebral congestion. Brain-tumors and continued epileptic attacks usually cause increased cerebral vascularity.

**Symptoms.**—*Acute cerebral congestion* of a marked degree usually causes intense throbbing headache, haziness or blurring of vision, and sometimes photophobia. It sometimes leads to vertigo, ringing or throbbing in the ears, a tendency to somnolence, and if sleep ensues, frightful dreams are likely to disturb it. There are mental depression and incapacity for thought. There is increased arterial tension.

Acute cerebral congestion in a *grecé form* occasionally comes on like an apoplectic stroke and furnishes an *apoplectic equivalent*. The patient falls inert, unconscious, with stertorous respiration, relaxation of the sphincters, and may die. He usually recovers, however, in a day or two. Slight paralytic symptoms last several days longer and eventually completely disappear. All gradations of cerebral congestive attacks are encountered, and, as above indicated, it is often difficult to distinguish at what point they become pathological.

In many cases and in various chronic intoxications there is a tendency in some to delirium, in others to convulsive manifestations, during the attack of cerebral hyperæmia. These motor and mental excesses may reach epileptoid and unimised stages.

*Chronic hyperæmia* of the brain is usually a part of organic disease of that organ and does not here call for special mention, as its symptomatic value is commonly apparent. When it gives rise to symptom groups it is by exacerbation in the form of acute attacks.

The diagnosis of cerebral congestion is commonly easy. The feeling of fullness in the head, the injected eyes, the flushed face, full arteries, quickened pulse, contracted pupils, and the increase of all symptoms when the head is lowered are sufficiently striking. It is, often very difficult, however, to distinguish the epileptoid and apoplectiform attacks from those of pure epilepsy and gross brain-lesions respectively. In the case of apoplectiform attacks in plethoric individuals before the age of fifty, especially if they are alcoholic or gouty, the presumption is in favor of cerebral congestion and reservation of opinion is in order. In epileptiform attacks the history of the case will almost invariably illuminate the situation.

The symptom, hyperæmia, having been deciphered, it remains to trace it to its proper source, and to this treatment is directed. Repeated cerebral congestive attacks in elderly persons foreshadow cerebral hemorrhage and softening. In parietic dysentery they are likely to be followed by apoplectiform attacks and a rapid downward course. In other instances their significance is strictly related to their cause.

## CEREBRAL ARTERITIS.

Immense importance attaches to disease of the cerebral arteries, not so much on their own account as because of the dire consequences of hemorrhage and softening which they may entail.

**Acute arteritis** in the brain may follow infectious diseases, such as typhoid, variola, diphtheria, scarlet fever, measles, and puerperal infection. All the arterial tunics are involved, but distinct symptoms do not arise unless thrombosis ensues. This is particularly liable to occur, as the intima is often much thickened. The arterial inflammation in many instances arises first in the vasa vasorum, and is thence propagated to the arterial trunks. The softening that ensues may, according to Turner, be followed by cerebral hemorrhage.

**Peri-arteritis** is a descriptive term applied to a proliferating affection involving the external arterial coat. It is attended by diffuse or circumscribed thickenings and connective-tissue increase. The perivascular sheaths of the cerebral vessels become choked. This, taken with the weakened arterial wall, favors the formation of saccular dilatations. In many instances the cerebral vessels become beaded with minute aneurysms, which are prone to develop at the branching points. In themselves these solitary aneurysms favor rupture, and the fatty degeneration of the arterial and capillary walls increases this danger. As a fact, they are the almost invariable source of cerebral hemorrhage, and can usually be found by carefully washing out the clot.

Peri-arteritis is frequently caused by, or at least associated with, Bright's disease. It may be induced by tubercular infection, which usually invades the cranium by the arterial route. It is a concomitant of the involution of advanced years and of arteriosclerosis. Syphilis may produce it, as may gout, rheumatism, and alcohol. Symptoms are very vague and indefinite, or absent, until hemorrhage or infarction gives rise to plastic disturbances. The condition should be suspected in chronic Bright's disease and in cases showing arterial degeneration elsewhere.

**Chronic arteritis, atherosclerosis, endarteritis deformans**, is frequently found in the large cerebral vessels, particularly those at the base, and especially the basilar. The atheromatous plaque originates in the occlusion or inflammation of the nutrient artery, or vasa vasorum. This produces an infarct largely confined to the middle tunic of the vessel, and the fatty degeneration that ensues is eventually replaced by calcareous deposits. The vessel may be completely encircled by such a patch, or numerous atheromatous islands may be found. By their coalescence the entire artery becomes rigid and brittle. At first the intima covers the plaques smoothly, but it is prone to break down, leaving the calcareous matter exposed in the blood-stream. This frequently leads to local deposits of fibrin from the blood, which may cause thrombotic closure of the vessel or wash away in embolic masses or particles, to produce disturbance farther along the course of the arterial current. In the same way calcareous particles may be cast into the stream, and, lodging in the narrowing channel, cause secondary mischief. Another effect of atheroma is to narrow the lumen of the affected vessel



through the thickening of its walls and the swelling of the internal coat. Again, by weakening the vessel-wall, atheroma may produce dilatation and lead to an aneurysm. The small arteries arising at the seat of atheromatous invasion may be occluded, though the parent vessel remains pervious. Collateral territories are thus cut off, while the ultimate distribution remains active. The rigidity and brittleness of the artery favor rupture and hemorrhage.

Atheroma may be considered as a purely senile condition in many cases, a part of the involutionary changes of the organism in advanced years. There is no doubt that gout, rheumatism, great muscular straining, overindulgence in alcohol, lead poisoning, and syphilis are additional causes. Lancereaux says chronic malarial infection may cause atheroma. Causes are frequently combined in a given case, as gout and senility. Atheroma, though usually found after middle life, has been noted in the aorta and large systemic vessels in children and even in infants.

The symptoms of atheroma of the cerebral vessels are usually vague and uncertain until thrombosis, aneurysm, or hemorrhage has been added. It may be reasonably suspected when the condition in the heart, aorta, and palpable systemic arteries indicates its generalized distribution. Prolonged nosebleed after middle life is generally due to arterial degeneration of carotid branches in the nasal spaces and a rather common forerunner of cerebral arterial accident.<sup>7</sup> Lesion of the optic chiasm by bilateral atheromatous thickenings of the carotids pressing upon it has been noted. Double temporal hemianopia may thus be produced. The formation of an aneurysmal tumor gives rise to its own localizing symptoms. Resulting hemorrhage and thrombosis present symptoms related to the structures that are injured or destroyed.

The treatment of atheroma is practically the same as that of arteriosclerosis.

**Arteriosclerosis, arterio-capillary fibrosis,** is always a generalized systemic condition, but it may be more accentuated in certain bodily organs and there give rise to local symptom groups. Its effect upon cerebral activity is most important. According to Sanson, the changes brought about are due to a poison circulating in the blood, which acts upon the fibroid elements of various tissues, but preëminently upon those of the arterial channels. The essential histological modification consists of a fibroid proliferation or fibrosis. In certain locations this acts mechanically to strangle associated structures, as, for instance, in the arteries, where the muscular fibers are thus invaded and even displaced. The walls of the entire arterial system become thickened. This may be due: (1) To thickening of the internal coat, which may go on to the complete obliteration of small vessels, or (2), commencing first in the external wall, the disease may spread inward, usually causing at first some hypertrophy of the muscular coat, or (3) the fibrosis may originate outside of the arterioles, which are involved secondarily by extension of the process to them. The various initial locations of the disease seem to depend upon the mode of the poisonous invasion. In one instance it affects the intima directly from the blood-stream, in

<sup>7</sup> K. Koops, "Arch. f. Laryngol.," 1899.

another the outer coat from the perivascular or lymphatic space, and in the third variety the fibrous structures of parenchymatous organs are disturbed through the lymph-channels. All these varieties may be found in the same case.

This condition has a number of pathological associations. Atheroma is present in about one-half the cases. Cardiac hypertrophy and dilation, hepatic cirrhosis, Bright's disease, asthma, angina pectoris, and mitral stenosis are frequently associated and due to identical changes. The effect of arteriosclerosis is to diminish the arterial caliber and thereby lessen nutrition. This may reach a complete degree and in the brain give rise to localized anemia and softening.

The nature of the poison which stimulates the fibroid activity is obscure. Leonis says the "general fibrosis has its origin in a fibroid diathesis either hereditary or acquired," but this explains nothing. By some writers defective elimination, particularly that from the kidney, is accused. Arteriosclerosis is certainly an accompaniment of old age and is a fair index of the wear and tear the individual has undergone and of

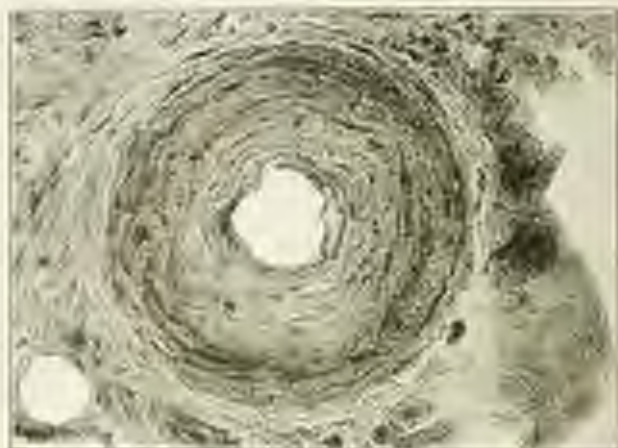


Fig. 34. Arteriosclerosis: thickening of all the coats (Delahanty).

the remaining vital capacity. We find it often a marked family characteristic. It is also clear that chronic intoxications by alcohol, lead, gonorrhea, rheumatism, and syphilis favor it strongly. Overeating, repeated muscular strains, and intestinal, renal, muscular, and cutaneous sluggishness count for something in its causation.

The cerebral symptoms produced by arteriosclerosis cover a wide range, but are all due to faults of brain-nutrition. They embrace those of senility, premature senility, and degenerative processes, both chronic and acute. The highest and most delicate brain-functions are likely to be first affected. We, therefore, find lessened mentality, aphasia, and monoplegias or mere clumsiness of the hands. Paresthesias are very common. Hemiplegia and diplegia manifestations are observed. At first these symptoms are temporary and recurrent, but unless the arterial condition improves, they tend to become more and more continuous



and permanent. Slight attacks of lethargy or sleepiness may eventuate in stupor, coma, and even in death. Jacksonian and generalized convulsions, syncopic attacks, and periods of mental confusion are all within the range of this protean malady. If the fibrin change occludes a cerebral vessel, it acts like a thrombus, to which, indeed, it often leads, and a softened infarct results in permanent loss of localized brain-function. Many islands of softening and many sclerotic patches may be due to this cause and present multiple symptoms. Diffuse sclerotic processes in the cortex are associated with it, and it underlies some of the cerebral lesions of general paresis and tabes. Associated minor symptoms, such as vertigo, headache, insomnia, irritability, lack of mental energy and muscular force, and the craving for stimulants, all point to the lowered nutrition of the brain. All the manifestations of cerebral arteriosclerosis are likely to come at first in gusts and waves.

**Symptoms.**—The physical examination of a case of arteriosclerosis usually demonstrates a tortuous, rigid, frontal artery, sometimes moving under the skin in a vermicular manner at each pulse-wave. The cornea commonly is the seat of a marked senile arcus. The radials, brachials, femorals, and all palpable arterial trunks are tense and rigid. The pulse is one of high tension. A light finger imperfectly detects it, but it seems to increase as pressure is applied and van hardly be obliterated. The sphygmomanometer gives abnormally high readings. There is usually an enlarged, laboring heart, and often roughened valvular sounds. The second sound is invariably accentuated. The urine is likely to show albumin and the formed elements that mark chronic nephritis. Often the quantity of urea is scanty or markedly deficient. Cirrhosis of the liver is common. A constipated habit is the rule, and the general health is below par. In less advanced cases the general indications of the arterial state may be very slight and yet the cerebral mischief may be extensive. This is especially true in the syphilitic varieties. The same processes that take place in cerebral structures affect the cord, and may, and often do, give rise to organic mischief, furnishing the basis of many systematized and unsystematized cord-lesions.

The treatment of arteriosclerosis affecting the brain must be undertaken at an early stage if much is to be accomplished. Toxic causes, if present, must be eliminated. Syphilis, gout, lead poisoning, alcoholism, renal, pulmonary, and cardiac conditions must receive their appropriate management. The arterial spasm due to the local irritation of the muscular tube, and perhaps also to uric products in the circulation, must be overcome. To relieve the spasm the nitrites, especially nitroglycerin, may be given at short intervals. Mercurials and much drinking water to cleanse the intestines and stimulate the kidneys are valuable aids. Alkaline waters such as Vichy and the lithia waters are good. Unless the water is freely excreted it will cause an increase of arterial pressure which is to be avoided. Care of the digestive tract and of the diet is of the first importance. All excesses must cease. A simple, easily digested regimen, with a very limited amount of red meats, starches, and sugars, should be ordered. The skin should be kept active by baths, frictions, and massage. If muscular exercises are for any reason contraindicated or not available, massage may take

their place. An outdoor habit should be cultivated, and change of scene may do much to reduce the mental distress and vague broodings. Of all medicine directed toward improving the arterial condition, the iodids easily hold first rank, but it should be remembered that the potassium salts depress the heart's action and perhaps add to the arterial over-tension. Sodium iodid is much to be preferred and is usually better tolerated. This should be given in doses of from five to twenty grains after meals, and continued for months and years, with short intervals. A good plan is to order the iodid discontinued during every fifth week. Tonics are almost invariably required. Arsenic can be readily given with the iodid, and strychnin is perhaps the best aid to the laboring heart. Avoid digitalis and everything else that tends to increase arterial tension. No harm seems to come from the frequent use of amyl nitrite or trinitrin, and the prompt, though transient, relief produced is often very gratifying, besides in a way confirming the diagnosis.

Under such a plan of treatment, aphasias, mental disturbance, hemiparesis, and many other symptoms of brain disturbance will some times rapidly clear up and, if not cured, remain in abeyance for years, provided moderation in all things be the rule of life. The prognosis, however, should be guarded, as we know that brain-cells degenerate beyond recovery if entirely deprived of their blood-supply for a few hours. At best it is evident that the presence of arteriosclerosis signifies a shortened life-span.

**Syphilitic Arteritis.**—It is now generally recognized that cerebral arteritis from syphilitic infection may be a comparatively early manifestation of the disease. Ogilvie shows from Nannva's statistics that syphilitic diseases of the cerebrospinal axis present the greatest proportion of cases during the first year following the initial lesion, but they may appear even after a series of years. The basilar, carotids, circle of Willis, and large cerebral arteries are those usually implicated, but smaller brain-arteries may be similarly diseased. The specific inflammation may produce a peri-arteritis and nodular plaques that look something like those of atheroma, or it may invade and infiltrate all the arterial walls with gummy products, commencing either as an endarteritis or a peri-arteritis. Long-standing syphilitic arteritis, especially of the large vessels of the base, produces a sclerous degeneration that does not cure. It is generally circumscribed in small patches, causing bulging of the internal and external coats, deforming the artery and altering its capacity.

Syphilitic arteritis leads to: (1) Obstruction of the vessel by the production of thrombosis or by an obliterating endarteritis; (2) rupture and hemorrhage, and (3) aneurysm. It may appear at any age and may follow inherited syphilis in children and even in adults.

The most prominent indications of syphilitic disease of the cerebral arteries are the prodromata. Of these the syphilitic headache, coming on usually toward evening and lasting until midnight, is the most distinctive. Except that due to tumor, it is the most intense and unmanageable headache with which the physician is called upon to deal. Ordinarily it is not confined to any portion of the head, but is described as being somewhat superficial, unlike the deep-seated pain of tumor. Un-



less the condition is now recognized or, as is rarely the case, spontaneously subsides, disturbances in the cortex are likely to appear, marked by paresthesias and loss of power in the extremities or disturbance of speech and the special senses. After vacillating symptoms of this character, which have a tendency to recur at intervals of a few days or weeks, an apoplectic stroke, due to sudden rupture of the vessel, may ensue, or a complete thrombosis lead to cerebral softening. When hemorrhage or thrombosis takes place the headache usually disappears, or sometimes it disappears a few days before the onset of serious results. The symptoms are those of cortical irritation and the eventual onset of paralysis is usually not marked by complete loss of consciousness, except in the hemorrhagic form, and coma is the great exception. Aphasia, facial paralysis, monoplegia, parasthetic tinglings, preceded by a history of violent headache, with nocturnal exacerbations, strongly indicate syphilitic disease of the cerebral arteries, even in the absence of any history of specific infection. As has been shown by Charcot, almost invariably there is some degree of basilar syphilitic meningitis in these cases and transient or permanent disturbance of the ocular apparatus is often added.

According to Charrier and Klippel, the chief groups of cerebral manifestations of syphilitic disease of the arteries are: (1) Apoplexy; (2) paralysis from obliterating arterial disease; (3) slight apoplexia and transitory varying palsies, and (4) intellectual disturbance somewhat similar to that of general paresis. Syphilis is the basis of true general paresis.

The treatment of the condition should be energetic even when it is diagnosed early, and will be discussed at length under the consideration of General Syphilitic Diseases of the Nervous System.

**Acute arterial degenerations** of an amyloid and fatty character affecting the cerebral vessels may follow numerous states marked by various systemic infections. Only in very rare instances do they give rise to marked cerebral symptoms, and these are usually overshadowed by the general state. Rupture and hemorrhage or thrombosis and softening may be due to them.

## CHAPTER VI.

### CEREBRAL HEMORRHAGE AND THE HEMIPLEGIC STATE.

EXCEPTING traumatic cases, hemorrhage into the substance of the brain is a secondary or terminal effect of degenerative or inflammatory disease of the cerebral blood-vessels, almost invariably of the arteries. Usually of comparatively slight seriousness in itself, the resulting injury or destruction of important brain-structures entails permanent disability if an early fatality is escaped. All parts of the cerebrum are subject to arterial disease and resulting hemorrhage, but certain locations present special liabilities. The most frequent site is in the distribution of the lenticulostriate arteries arising directly from the

trunk of the middle cerebral and supplying the basal ganglia and their internal and external capsules. Of these, the anterior branch is the so-called artery of cerebral hemorrhage. After the region of the basal ganglia and capsules, in order of frequency as sites of cerebral hemorrhage, follow the centrum ovale, cortex, cerebellum, pons, medulla, crura, and corpus callosum. So commonly, however, does hemorrhage occur in the neighborhood of the large ganglia that a somewhat definite clinical type of cerebral hemorrhage is presented. This will first engage our attention, and then the less frequent sorts and varieties, all of which have symptoms in common, may be rapidly sketched.

**Pathological Anatomy.**—Cerebral hemorrhages of the vesicular variety occurs with about equal frequency on the two sides of the brain. The quantity of blood extravasated depends upon two factors, (1) the size of the blood-vessel and (2) the arterial pressure, but the pressure is much the more important of the two. According to Wernicke "the apoplectic or traumatic effect is equal to the product of the amount of effused blood into the square of the pressure with which it is extravasated." Ordinarily the hemorrhage commences in the anterior portion of the lenticular nucleus and separates or tears through the adjoining structures, involving the internal capsule, the external capsule, and the optic thalamus. It may extend upward into the centrum ovale or tear into the lateral ventricle, and finally break through into the pial spaces at the base near the optic chiasm. Very exceptionally the blood breaks through the cortex of the convexity, but commonly it is arrested at the lower surface of the gray matter.

Recent hemorrhages show a coagulum bathed in serum and non-adherent to the surface of the hemorrhagic pocket. After a few days the serum is relatively increased, the coagulum is contracted, and is attached to the adjoining structures by fibrous trabeculae. Absorption of the serum then follows. The clot is resorbed in part, and finally only an ochreous mass remains, made up of blood-crystals, pigment, and fatty detritus. By thickening and participation of the surrounding brain a sort of cyst is formed. If it is small and its walls compact, cicatrization may ensue. About large so-called apoplectic cysts there is usually a zone of degeneration and fatty softening. This is often the seat of minute hemorrhages, and is likely to undergo inflammatory changes or by infection become purulent. An abscess thus results in which, sometimes, the partially organized hemorrhagic clot floats.

Even in recent hemorrhages there is difficulty in determining the origin of the blood. By carefully washing away the clot under water military anemysms are almost certain to be found. They lead the arterial vessels that are brought to light, and sometimes one is fortunate enough to find the minute one originally ruptured and still containing a fragment of organized clot. Other vascular lesions that occasionally give rise to cerebral hemorrhage are amyloid, hyaline, syphilitic, and fatty arterial degenerations and acute infective arteritis. Not infrequently cerebral hemorrhage is secondary to a softening through which the artery is robbed of its proper support and nutrition. In ancient cases cicatrization and spreading degenerative changes usually obscure the exact vascular fault.



The motor tracts that are cut by the lesion present a descending degeneration which extends downward the whole length of the neuron. In the cord the pyramidal tracts are usually both involved, but to a greater extent on the side opposite the lesion. Exceptionally in cases of long standing, changes in the upper motor tract entail secondary disturbances on the lower motor and trophic neuron, and an amyotrophic condition is superadded, with corresponding degeneration in the anterior horns.

**Etiology.**—In any given case of cerebral hemorrhage there are, ordinarily, a number of causative elements. Almost invariably there are (1) *high arterial tension* and (2) *lowered arterial resistance*. The causes of ordinary aneurysm are much the same as those of rigid arteries, atheroma, and aortic hypertrophy. The strongly acting heart drives the blood-column through the rigid aorta and carotid, and its full force falls upon the arteries arising from the circle of Willis. These are of comparatively small caliber, and, not having outlets by anastomosis, oppose a dead wall of resistance to the directly received aortic impulse. Surrounded in turn by perivascular spaces and not supported by firm parenchymatous tissues, their walls weakened by age or infection and yielding at numerous points to the formation of saccular dilations, arterial rupture naturally follows.

The predisposing causes of cerebral hemorrhage are those that produce or tend to produce the primal arterial disease. Advanced age, gout, alcoholism, rheumatism, plumbism, and syphilis are chronic states favoring it. Acute infections, tending to produce acute arteritis or amyloid and fatty degenerations, such as purpura infection, diphtheria, scarlet fever, etc., are possible active factors. Whooping-cough in children is an occasional cause, the factors of strain, infection and lowered resistance all being present. An undoubted *hereditary tendency* to cerebral hemorrhage is sometimes encountered. The writer is familiar with one family of eleven brothers and sisters, nine of whom have died from cerebral apoplexy. The so-called apoplectic habit has no significance if not associated with other predisposing conditions. The relation of age to cerebral hemorrhage is an important one. During the first year of life it is relatively frequent, and drops thence to the end of the first decade. Gowers states that a proportion of 1.8 to every 1000 under the age of ten have suffered from this accident. From ten to twenty-five it is infrequent, and then rapidly mounts, reaching its maximum at about fifty-five, thence again descending, and seldom occurs after seventy-five years of age. The male sex shows considerable preponderance, about three males to two females suffering from cerebral hemorrhage. This is due to their greater tendency to excesses and increased liability to exposures. Temperate climates and winter months show an excess of cases over the opposite conditions.

Everything which induces an accelerated circulation must be ranked as an exciting cause. All physical, mental, and moral shocks, and all intense emotions may lead to cerebral apoplexy in those predisposed. Muscular efforts, coughing, sneezing, straining at stool, coitus, and vomiting have induced it. Derangements of the circulation with cardiac effort, due to exposure to the cold or from a cold bath or a bath after a full meal, have served to rupture the diseased artery. In the Great Lakes

region of this country the frequent sudden changes in barometric pressure, attended as they very often are by extraordinary variations in atmospheric humidity and temperature, constitute a menace, if they do not furnish an exciting cause of cerebral hemorrhage. It often follows a delirium.

**Symptoms.**—The onset of cerebral hemorrhage of the capsular variety is almost invariably abrupt, and constitutes the type of cerebral apoplexy that is familiarly and properly called "a stroke." In very exceptional cases it is preceded by momentary confusion, vertigo, uneasiness, or other vague subjective disturbance. Most of the alleged *prodromata* are merely symptomatic of the arterial condition that always precedes the stroke, and are common to peri-arthritis and arteriosclerosis. A hypertrophied heart, rigid artery, and high arterial tension, a contracted kidney and albuminuria with or without formed kidney elements, are of some value in presaging an apoplectic seizure from cerebral hemorrhage. The patient, ordinarily, is stricken down, and in the fulminating cases may die instantly. If walking, he falls heavily, or if sitting, reels out of his chair. Consciousness is almost instantly abolished. The action of any of the exciting causes named above will correspondingly affect the opening scene. At first the face is pale, the pupils contracted, and unocular twittings or, rarely, active general convulsions may occur. There may be considerable motor restlessness and uneasiness until coma becomes profound.

In the apoplectic state that follows the patient lies inert, unconscious, breathing slowly and stertorously, and often presenting the Cheyne-Stokes respiration. The face is mottled or even deeply congested, covered with perspiration, and expressionless. The half-open eyes present sluggish or inactive and usually contracted pupils. The conjunctiva is usually congested and insensible, and the palpebral reflex is wanting. All forms of sensibility are abolished. The sphincters are relaxed and the limbs limp. There is usually urinary retention, and overflow occurs later. At this time it may be difficult to determine which hemisphere has been injured. Sometimes, by careful examination, the paralyzed side is found more completely relaxed than the other, in which, perhaps, some slight resistance to passive movements is present, or the sound limbs when raised do not fall so heavily. There is also, even from the first, a tendency for the patient to direct his face and eyes in *enversive deviation* to the side of the brain that contains the hemorrhage. This indicates muscular paralysis on the side from which they are turned. This deviation must not be of the spasmodic sort, which has an opposite significance. Uhthoff<sup>1</sup> has found a choked disc in eleven per cent. of cases of cerebral hemorrhage, optic neuritis in six and a half per cent., and retinal hemorrhage in nearly three per cent. At first all the cutaneous and muscle reflexes are temporarily increased, but immediately subside and tend to disappear. The superficial reflexes, as a rule, are soon abolished on the paralyzed side. The unilateral absence of abdominal, cremasteric, plantar, and conjunctival responses furnishes important information in the early hours of the attack. The toe-sign of Babinski almost invariably develops on the affected side

<sup>1</sup> "Neurolog. Centralbl.," No. 20, 1909, p. 1106.



within a few hours, even within a few minutes, and persists indefinitely. The comatose condition either deepens into death or passes off.

After a variable period of hours or even days the coma, in cases not immediately fatal, gradually yields to torpor, in which the patient can be partially roused by strong and especially by painful stimulation. He pushes away the pinching fingers of the examiner, mutters a little, takes a deep, quick inspiration, or otherwise manifests discomfort. Then he hears loud voice-sounds, and finally shows, by facial expression and attempts at speech, that he has regained partial consciousness. If undisturbed, he relapses into stertorous sleep, and the expired air pulls out the fleshy cheek of the paralyzed side of the face and escapes through the angle of the mouth on that side. Finally consciousness is restored.

Shortly after the stroke the rectal temperature is slightly lowered, but is likely to rise a little above normal after a day or two. In all cases after a few hours the temperature will be found higher on the side from which the eyes are averted—that is, on the paralyzed side—than on the other. This is true of the mouth, face, trunk, and extremities, and the difference amounts to from  $1^{\circ}$  to  $2^{\circ}$  F. At the same time this warmer, paralyzed side often shows increased perspiration, and usually an intensified redness and congestion as compared with the other.

The motor loss in this variety of cerebral hemorrhage is hemiplegic. After consciousness has partly returned its distribution can be plainly



Fig. 35.—Right hemiparesis with atonous face. 1, Ordinary expression; 2, spasmodic involuntary laughter, showing bilateral action of facial muscles.

determined. Usually the arm is much more affected than the leg, and the face shows great variations of implication in different cases. In some it is scarcely affected. As a rule, the distal portion of the limb is much more affected than the portion near the trunk. The hand thus suffers proportionately more than the arm or shoulder, the foot than the thigh. The muscles of the trunk, being paired and fully represented bilaterally in the cortex, do not show much one-sided loss of power. A careful examination will, however, detect reduced respiratory excursions in the paralyzed side in forced respiratory movements, and unilateral weakness in the acts of sneezing, coughing, etc.<sup>1</sup> The facial asymmetry offers several important and significant peculiarities. Except in the rarest instances it is only the lower half of the facial-nerve distribution that is much impaired. The frontal and orbicular regions show but little lack of power;

<sup>1</sup> Seward, "Arch. de Neurolog.," Dec., 1899.

the brows are raised, the eyes close and open nearly as well as ever, but a slight unilateral parietic defect is certainly present as a rule. In the lower face the lack of muscular power and tone allows the mouth, chin, and even the nose to deviate to the sound side. All voluntary attempts to use the labial, nasal, and zygomatic muscles increase the deformity, but usually in *emotional* expression, as in laughing or weeping, the lack of symmetry tends to disappear (Fig. 85). Only when the basal ganglia, and particularly the thalamus, are seriously invaded by the hemorrhage do emotional expressions also intensify the one-sidedness. Even then the retention of power in the upper half of the face distinguishes this palsy from that of disease of the seventh nerve. The conjugate deviation of head and eyes passes away as consciousness returns, but the tongue when protruded usually turns strongly to the paralyzed side, owing to the weakness in the corresponding genioglossus. With the tongue protruded the patient cannot move its tip across the median line toward the sound side nor thrust it so strongly into the sound cheek. A deviation of the tongue toward the affected side is not a rare occurrence. Jones<sup>1</sup> has found it to be the case in over 10 per cent. of hemiplegies. This lack of muscular power in the tongue and lips tends to muffle and thicken the speech, which may even become nasal, as the soft palate shares in the muscular weakening and droops on the paralyzed side. Aphonia is not ordinarily produced by capsular hemorrhage. In exceptional cases the lesion implicates the corticopeduncular speech-tracts, producing a variety of motor aphasia marked largely by ataxic control of the muscles of speech-production.

**Sensory Disturbances.**—As consciousness returns after the stroke, ordinarily the complete cutaneous insensibility disappears. Only in those cases in which the lesion destroys the sensory pathway in the posterior third of the posterior limb of the capsule do we have persistent *hemianesthesia* corresponding to the hemiplegia. This identical anesthetic field is sometimes encountered in hysteria, which may indeed be induced by the shock of a cerebral hemorrhage as well as by any other accident, and may give rise to a perplexing combination. Close scrutiny for other evidence of antecedent hysteria should be made in all such cases. The sensory disturbances in the limbs is usually more pronounced toward their distal extremities, where the paralysis is also always most marked and persistent. *Hemianopia* is frequently present immediately after the stroke, but usually passes away in a few days with the other sensory disturbances. When the visual path at the sensory crossway is injured or the lesion is in the occipital apex, hemianopia persists. Even when the patient is still inclined to stupor it may be detected by bringing the fingers into the visual field first from one and then from the opposite side. The ophthalmoscope frequently detects an engorged papilla due to the increased endocranial pressure. Disturbance of other special senses is subject to the same rule. Often there is considerable pain and sensitiveness in the paralyzed limbs. Severe headaches and vertigo are uncommon until the hemiplegic state is established, and even then they are rare.

Trophic disturbance in the early days after cerebral hemorrhage is rare. An *uride balane* sometimes appears over the buttock of the

<sup>1</sup> *Jour. Nervous and Mental Dis.*, Oct., 1911.



paralyzed side, and in a few days, almost in a few hours, attains large dimensions. Such cases almost invariably soon terminate in death. Rapid calcification and even rapid following are encountered at times. Disturbance of the medullary nuclei probably accounts for temporary albuminuria and glycosuria, which are incidentally observed in some cases. To the same source some attribute the rare gastric and intestinal *hemorrhages*. Occasionally a large joint on the palsied side, notably the shoulder, may develop an acute arthritis, and all the articulations of the paralyzed limbs are later on inclined to rigidity out of proportion to their disease. After the first week a rapid elevation of temperature is sometimes produced by the development of an infectious cerebritis about the hemorrhagic focus. It usually ends fatally.

The hemiplegic state finds its prototype in the cases that survive the stroke of cerebral hemorrhage. It must not be overlooked that it is a sequency common to many cerebral and spinal lesions, and is associated with some neuroses. It may follow: (1) Traumatic lesions of the brain and cord; (2) meningeal lesions due to hemorrhage, inflammation, syphilis, and tuberculosis; (3) cerebral lesions due to hemorrhage, softening, tumors, abscesses, and sclerosis; (4) cerebro-spinal lesions of tabes, multiple sclerosis, and general paralysis of the insane. It may be due originally to: (1) Intoxication by uremia, diabetes, alcohol, lead, mercury, and some carbon compounds; (2) infections from pneumonia, malaria, typhoid fever, purpural fever, eruptive fevers, diphtheria, influenza, syphilis, and tuberculosis; (3) it marks some cases of chorea, hysteria, and paralysis agitans.

In cerebral hemorrhage, if death does not occur during the first three weeks, improvement in the paralytic features uniformly appears and is progressive for several months. Finally, all gradations are encountered, from the slightest paresis to complete hemiparalysis. In a well-marked case, in which at first the hemiplegia is complete, we may expect some return of voluntary motion after ten or fifteen days. The lower extremity first shows improvement, and is followed by the face and later by the upper extremity. In six to eight weeks the patient may stand and perhaps with much aid walk a little. During this time a notable change has appeared in the reflexes and the tone of the paralyzed muscles is greatly altered. At first the reflexes are abolished or greatly diminished and the muscles hang limp and flabby from the tones. Gradually the reflexes increase and become exaggerated. Transient stiffness and rigidity are then found in the limbs. Ankle-clonus, rectus-clonus, and even wrist-clonus appear, and with the temporary rigidities fore-shadow the later contractures. The increase in myotatic irritability may be expected to appear during the third week, and is always present sooner or later, if the motor pathway in the brain is injured by the hemorrhage.

Introduced by the myotatic irritability, clonus, and the fleeting attacks of rigidity, the period of late contractures is established from one to four months after the stroke. In very rare cases it is never fully developed, and in other rare cases, owing to secondary changes in the trophic centers of the cord, it disappears after being well marked for years and muscular atrophy develops, but the rule is that once present it is progressive for a time and then permanently remains. Under the action

of this contracture the limbs assume characteristic rigid attitudes that constitute great deformities. It sometimes strongly involves the face, which is then drawn to the affected side and gives to the sound side a false appearance of weakness.

In the *upper extremity* the flexors predominate to draw the digits into the palm of the hand, to flex the wrist, to pronate the forearm, and to fix the elbow at an angle. The extremity is usually held close to the chest. In these vicious positions the joints become practically soldered. After the case is somewhat chronic, if the fingers or wrist are extended, they fairly snap back to their flexed positions, and the tightly clenched fingers may cause trophic lesions in the palm. In cases of long standing, probably both from the enforced inactivity and trophic disturbance, the range of joint movements is considerably limited irrespective of the muscular contracture, and sometimes this appears early.



Fig. 55.—Contractured positions in hemiplegic hand.

In the *lower extremity* extension prevails. The knee is held quite rigidly extended and there is a tendency to equinovarus, so that the foot rolls over on its outer border. These are the usual contractures, and give to



Fig. 56.—The hemiplegic gait. 1 and 2, Advancing the hemiplegic leg in a clank from the hip; 3, bearing weight on parietal leg and stance while advancing sound limb.

the patient an *attitude* and a *gait* that are distinctive. It will be recalled that the distal portions of the extremities are most affected, and



now, rigidly fixed by the contractures, they are moved *in vacuo* by the muscles of the pelvic and shoulder girdles. In walking the rigid lower extremity is used somewhat as a peg-leg. The body-weight is carried on the sound limb, over which the trunk inclines outwardly, and by body and pelvic movements the paretic lower limb is swung forward, the dragging toe describing an arc-like course around the heel of the sound side. Then upon the paralyzed limb, often trembling with clonic action, as upon a staff, the next step is taken by the sound side, aided usually by a cane or crutch. When in the dorsal decubitus, the patient cannot lift the extended sound leg from the bed, though he may be able to lift the heel on the paralyzed side. This is due to the inability to fix the pelvis and the lower extremity on the affected side, which is necessary to raising the opposite limb.

A marked minority of cases show a type of extension in the upper extremity, which is most pronounced at the elbow. The limb is held rigidly by the side of the body. The wrist and fingers may, though very rarely, also be extended, and the supinators may overbalance the pronators. Again, flexion may predominate in the lower limb, or extension may be present in the upper and flexion in the lower limb, or different segments of the same limb may show opposite conditions as to flexion and extension. This but rigidity may render it impossible to demonstrate the *muscle reflexes*, which, nevertheless, are greatly exaggerated. The *tee-sign* is usually present and readily elicited. Like the myotatic irritability, the late contractures are due to the descending degenerative processes in the motor tracts.

The paralyzed limbs show marked *circulatory disturbance*. They are at first reddened and cyanosed. The blanched spot caused by finger-pressure only slowly disappears. Sometimes they are sodden with perspiration and show epithelial variations, either by increase or more often by decrease of growth in the nails, hairs, and epidermis. They are cold later on, and their temperature is reduced several degrees as compared with the sound side. At times there is marked edema, which may come on very early in the case, and is attributable to the muscular inactivity, with lymph-stasis, and frequently is associated with a kidney or cardiac lesion.

A phenomenon is often noticed by hemiplegics that gives rise to false hope of returning voluntary movement in the paralyzed limbs. Sometimes without volition the paralytic hand may open or the leg draw up. These movements are perhaps analogous to the constant *athetoid features* that develop in some cases. The entire upper extremity, over which the patient has practically no control, may execute such movements as rising itself over the head in conjunction with the other arm in yawning, stretching, or other instinctive act. These associated movements probably result from the bilaterality of their cortical representation. Again, if the hemiplegic attempts to write with the sound hand, provided it is the one not formerly used for that purpose, the paralyzed hand may sometimes be seen to duplicate the motions of the one voluntarily used. Right-sided hemiplegics who are at the same time right-handed, in attempting to write with the left hand sometimes produce *mirror writing*, which is practically the normal method for left-handed individuals.

On the sound side there are also marked disturbances. The reflexes are exaggerated and even contracture may develop. The muscular strength is reduced from 10 to 50 per cent. Incoordination may be pronounced.

**Complications.**—The hemiplegic state often presents sensory complications. Subjectively these consist in paresthesias, such as feelings of heaviness, dragging, and formication. Sometimes the limbs seem to the patient to be entirely gone; others complain of phantom or additional limbs. According to von Bechterew,<sup>2</sup> lesions in the neighborhood of the lenticular nucleus are prone to produce this symptom, which he names *pseudomelia paresthetica*. Objectively we may have hemianesthesia if the sensory portion of the capsule is injured, and the other sensory disturbances of injury to the great crossway may be added.

A wide range of *posthemiplegic motor complications* are encountered. We find rhythmical tremors, as in hemiparkinsonism agens, or the intentional variety of tremor, as in multiple sclerosis of limited distribution. Irregular movements, like those of chorea, ataxia, and athetosis, are quite common on the affected side. When they result from bilateral cerebral lesions, great diagnostic difficulties are presented. The production of all these posthemiplegic motor symptoms requires that the paralysis should not be complete, and their presence implies a condition of irritation somewhere in the path of the upper motor neuron. This is usually furnished by lesions in the region of the basal ganglia, especially those affecting the optic thalamus and impinging upon the capsular fibers.

Among the *trophic complications*, in addition to the early, acute bed-sore, the paralyzed side may present indolent ulcerations over the sacrum, elbow, or heel. The *angiotrophic* disturbance already mentioned may come on within a few days of the stroke, or may appear late in the hemiplegic history, and, in either event, must be attributed to interference with the trophic control of the anterior horns. The electrical reactions then vary from those of simple quantitative changes to the reaction of degeneration. This muscular wasting is most common in the upper extremity, and, when appearing early in the case, is of ominous import. The skin, hair, nails, and fatty dermal layers may be increased or more rarely diminished in the paralyzed limbs.

It would also seem that dynamic, if not actual, changes in the anterior horns must be accountable for the occasional acute *hypotrophic necrosis* that develops in the paralyzed limbs in the early days after the illness, and for the acute *orthopedic affections* that also appear at that time. These are sometimes associated. The diseased joints suggest rheumatism. They are inflamed, painful, hot. The joint-surfaces and synovial organs rapidly disintegrate, and present practically the same condition as the arthropathies of tabes. When appearing early this joint affection is of bad import, like the acute decubitus and early angiotrophy.

Hemiplegia from the ordinary capsular hemorrhage does not necessarily disturb the mind. In the old it may precipitate the mental deterioration of senility, and it may be followed by organic dementia.

The course of cerebral hemorrhage of the common type may present many variations. Death may occur almost instantly, but this is exceptional, and usually the result of inundation of the ventricles.

<sup>2</sup> "Neurolog. Centralbl.," Sept. 1, 1903.



Death may occur during the coma from the great size of the clot and the exhaustion of the patient. After coma has disappeared and before contracture has developed, death may result from cerebritis arising from infection of the clot. During this early period pneumonia, often caused by the aspiration of food or drink, sometimes carries off the patient. A succession of hemorrhages taking place at short intervals, one apoplectic phase succeeding another, are nearly always fatal. After the initial stroke, and usually before the termination of the coma, there may be a sudden increase in all the symptoms. The breathing becomes rapid, irregular, stertorous, the unconsciousness more profound. The reflexes, if partly present, are entirely lost, and tetanic spasms or convulsions appear on the paralyzed side. The temperature bounds upward and the case terminates fatally in a few hours, or in a day or two. These symptoms are due to the hemorrhage bursting into the ventricles. If the fourth ventricle is invaded, nystagmus appears and death promptly follows. In the so-called *insystemant* apoplexy the hemorrhage begins without marked stroke, and, steadily continuing, produces the apoplectic state in the course of a day or two, finally inundating the ventricles and terminating fatally.

Recoveries from cerebral hemorrhage are not rare, and in the majority of cases the hemiplegic state is reached and the stage of contractures is developed. Very exceptionally the hemiplegia practically disappears. This is only possible when the hemorrhage has affected the capsular tracts by pressure without producing rupture of their fibers, and then the clot is necessarily small. As the greatest rarity the recovery may be so complete that no trace of the former palsy can be discovered by clinical examination.

**Clinical Forms.**—Cerebral hemorrhage presents a number of clinical forms, the most usual of which has been the basis of the preceding description. Variations in localization and in the amount of extravasated blood endlessly modify the type. Within the field of the lenticulostriate arteries a very small hemorrhage may produce a partial hemiplegia or a monoplegia, though this is a rare form. We have the occasional form of hemiplegia with hemichorea, or hemitaxia, or hemiatetosis. There is the form of hemiplegia with hemianesthesia and another in which hemichorea and hemianesthesia are combined. A rare form consists of a facial monoplegia with hemianesthesia, and a form presenting hemianesthesia and motor aphasia has been noted. Hemorrhage into the posterior lobes of the brain is attended by the apoplectic onset or insult and leaves visual or optic remnants if it involves the corresponding radiations and pathways. In the anterior lobes and in a large portion of the centrum ovale a considerable hemorrhage may take place without producing lasting symptoms. In these hemorrhages remnants from the capsule hemiplegic symptoms are sometimes present at first and then disappear as the traumatic effect of the apoplexy subsides. Primary cortical hemorrhage is exceptionally encountered. The clot is usually small, owing to the small caliber of the cortical arteries; the stroke, therefore, is slight or absent, but stupor and semicomat may be present. The symptoms produced are those of an irritant lesion. If it occurs in the motor zone, repeated limited convulsions of a Jacksonian

type are likely to occur and may become generalized. Hemorrhages into the pons and medulla, if not immediately fatal, give rise to localizing symptoms and cranial-nerve disorders that have already been discussed, including the numerous crossed paralyses.

**Cerebellar hemorrhage** usually presents a very sudden onset, but there is correspondingly much less disturbance of the mind and consciousness than occurs in the cerebral variety. The patient falls, vomits, and experiences intense vertigo. Attempts to sit or stand greatly increase the vertigo and vomiting and may render the horizontal attitude obligatory. If the lesion approaches the middle peduncle, forced movements or rigid positions may be induced. These may impel the patient strongly to the right or left, backward or forward, and in the reclining posture cause marked curving of the trunk in corresponding directions. Ataxia and asthenia in the extremities are immediately produced, and tetanoid spasms may appear. Sensory disturbances are fleeting or absent. Later on the preponderance of symptoms is on the side of the lesion if it is unilateral, but if pressure occurs on the pyramidal tracts above the decussation, crossed symptoms are presented. Cranial-nerve impairment is likely to be present from irritation of the nuclei and pressure on the floor of the fourth ventricle may induce dangerous pneumogastric complications. Should this ventricle be flooded, bulbar symptoms are induced and death promptly follows. Much similarity is presented by attacks of labyrinthian vertigo, and at first the diagnosis may be impossible. A history of preceding attacks and of ear disease is significant. If the cerebellar hemorrhage involves the auditory nerve, the two symptom groups coincide.

**The diagnosis of cerebral hemorrhage** is often difficult and sometimes impossible. Its consideration falls into two parts: (1) The diagnosis during the apoplectic state and (2) the diagnosis after the apoplectic state.

Shortly after the onset of the stroke we have to ask ourselves whether the case is one of syncope, poisoning, or alcoholic intoxication. If one-sided symptoms can be detected, all of these conditions may be excluded, and each has some distinctive symptom that one on his guard may detect. The pale face, fluttering pulse, and sighing respiration of syncope, the contracted pupil of opium, the smell of alcohol on the breath, etc., are suggestive, and taken with the history of the onset, when obtainable, are nearly sufficient. But cerebral hemorrhage may come on during drunkenness, or a hemiplegic case may be given stimulants. Uremia is more difficult to differentiate, and may occasionally counterfeit all the indications of cerebral apoplexy. Examination of the urine may throw light on the situation, but it is to be remembered that the conditions giving rise to uremia are those usually associated with arterial disease in the brain. Most uremic comatose cases, at some period, present vomiting, headache, motor excitement and sporadic restlessness, in which paralytic features are lacking. The coma is rarely profound at first. **Meningeal hemorrhage** may be easily mistaken for cerebral hemorrhage, as stupor, coma, convulsions, and paralysis are produced by both. The meningeal form, however, usually follows injury, and comes on slowly or after a distinct interval. It is often marked by irritation of cranial nerves and early bilateral excursions.



The *epileptic attack* usually has a history of anterior convulsions, presents clonic, tonic, and stuporous stages, and is quickly and completely recovered from. Attacks of focal epilepsy may indicate a cortical hemorrhage, but are common to all diseased conditions of the cerebral motor surface. Every case must be carefully analyzed, and often, even then, only a presumptive diagnosis can be finally reached. *Hysteria* in rare cases produces an imitation of cerebral hemorrhage that is extremely faithful to the type. The attack, however, usually occurs under circumstances of emotional and psychical disturbances indicative of hysteria. The face is rarely involved, Babinski's toe-sign is absent, and sensory stigmata are commonly present. The age and clinical history are also significant. In general *parietal apoplectiform seizures* are common, and the differential diagnosis during the attack may be very difficult. A history of mental confusion, childishness of conduct, forgetfulness, inconstancy upon the feet, and syphilitic infection would favor the parietic side of the question. Almost complete recovery from the stroke may then be expected in a majority of cases. Spinal puncture frequently gives issue to a uniformly tinted bloody fluid that practically proves the hemorrhagic nature of the attack.

After the early apoplectic symptoms have persisted many hours or have subsided, an organic lesion can usually be determined from the hemiparetic features. The important question regards the nature of the vascular accident. Is it rupture or occlusion that has taken place? Have we to do with hemorrhage or with infarction? Are we in the presence of extravasated blood with a tendency to encapsulation, or confronted by thrombosis, the precursor of softening? It is a question of importance, both as to treatment and prognosis. It is sometimes an unsolvable problem, but should never be neglected. At the end of the next chapter a differential table is given, to which attention is now directed. In a general way we may say that the following points favor a diagnosis of cerebral hemorrhage: Sudden onset, absence of syphilis, and endocarditis, the presence of strong cardiac action comparatively early, high arterial tension, marked coma and cyanosis, lowered rectal temperature and raised axillary temperature, convulsions involving the whole of one side, paralysis involving the whole of one side, early improvement in the paralysis most marked in the leg, lack of permanent sensory disturbances, the presence of complete hemianesthesia in the absence of hysteria, the development of postapoplectic tremor, and athetosis. In the early days of the apoplexy a retinal hemorrhage or blood in the spinal fluid offers very significant evidence.

The topical diagnosis must follow the general considerations laid down in the discussion of the subject of cerebral localization. Attention is again called to the fact that the above description is dominated by the clinical aspects of the usual capsular variety of cerebral hemorrhage.

**Prognosis.**—A cerebral hemorrhage is always of serious import, as it is a conclusive demonstration of wide-spread and threatening arterial disease in the encapulation. However slight, it implies the probability of a recurrence. About two-thirds of all the cases survive the first

attack, one-third the second, and very few the third. The size and location of the clot are important considerations. The prognosis is grave in proportion to the violence of the attack and the depth and duration of the coma. Coma lasting three days seldom ends in recovery. The appearance of Cheyne-Stokes respiration or indications of ventricular flooding practically mean death. The occurrence of convulsions is a serious feature. Pneumonia is almost invariably fatal. Acute bedsores and acute joint disease are usually followed by death. A recurrence of coma or a sudden elevation of temperature or repeated apoplectic features imply an early fatality.

Unless there is some improvement in the paralysis at the end of the second week, it is likely to be permanent. The appearance of contractures implies lasting functional loss wherever they develop. The state of contracture commences from twenty days to three or four months after the stroke. The shoulder and hip movements improve more than the knee and elbow; the ankle and wrist, toes and fingers, progressively decline in recuperative prospects. The lower extremity surpasses the upper both in point of time and extent of recovery. Considerable improvement may be anticipated during the first two or three months, and then much slighter progress to the end of the first year or eighteen months. Thereafter the case will be practically stationary. The duration of life among apoplectics is about five years, according to Dam, and, as he points out, a stroke may be a conservative measure, enforcing an inactivity that prevents arterial strain and thereby prolongs life.

**Treatment.**—If a positive diagnosis of cerebral hemorrhage is made, immediate active treatment should be instituted, but in doubtful cases a masterly inactivity—an armed neutrality—is the proper course; there are many who think it the only course. The important indication is to reduce arterial tension, to discount the pressure at the site of hemorrhage. When the case is seen immediately after the stroke the head should be raised, an ice-cap applied, and faithful carotid compression on both sides of the neck employed for forty minutes. Meanwhile the blood should be directed to the lower extremities by having them swathed in hot compresses and by the intelligent application of mustard. Two drops of emulsified croton oil serve to practically bleed the patient into his abdominal vessels and at the same time unload the bowels. Regarding restriction, the forces are still divided. In plethoric, congested, livid cases with strong cardiac action and high arterial pressure it may properly be used.

In most cases the hemorrhage reaches its maximum within three or four hours and the damage is done. Thereafter the indication for treatment is solely to prevent a recurrence, and that means to keep the arterial tension down. Quiet, warmth, liquid diet, if any, free bowels, cleanliness to prevent bedsores, the administration of cardiac sedatives; attention to the bladder, which is likely to distend and overflow; care to prevent aspiration pneumonia, from food, mucus, or a septic condition of the mouth, and readiness to meet recurrent hemorrhages or intransigent apoplexy by carotid pressure will answer the major requirement. Ligation of the carotid is not a well-established proceeding.



and carotid compression must be used with circumspection and intelligence. In the aged and atheromatous it may produce convulsions or serve to increase the coma. The effect in controlling the circulation may be observed in the usually prominent and throbbing temporalis and in the color of the face. Suggestions of early trephining and evacuating the clot have been made. If rupture of the motor path occurs directly the extravasation of blood takes place, further surgical laceration can scarcely improve matters. If purulent cerebritis develop in the clot, trephining to drain such a focus may reasonably be attempted in this otherwise fatal complication.

As soon as the apoplectic coma has passed away, gentle massage of the paralyzed side and exercise of all the paralyzed muscles by mild faradism should be instituted. The purpose should be clearly in mind to secure as much improvement during the first month as possible and to postpone to the utmost the appearance of contracture. A muscle that may feebly respond to volitional control, perhaps to the associated action of the sound hemisphere, is rendered perfectly useless by contracture. From the onset extremely gentle passive movements of all the joints to their full range should be employed every two or three hours. Every one is familiar with the rapidity with which muscular and joint stiffness appears in splinted extremities. In these hemiplegic cases the limbs are splinted by paralysis. As soon as there is any reappearance of voluntary motion the patient should be encouraged to exercise it. These measures are usually postponed to the end of ten days or a fortnight in fear that, by instituting them early, the brain-lesion may in some way be increased. Certainly any violent or severe measure is most strongly to be deprecated, but a common-sense application of the foregoing directions will be found to yield encouraging results.

When contractures appear, every effort should still be persistently made to counteract their deforming effect. We are familiar with the vicious attitudes they produce. In the upper extremity the extensors should be encouraged by massage, electricity, and forced passive movements to overcome the usual flexor supremacy. Similarly, in the lower extremity the equinovarus should be overcome, if possible, by stimulating the antero-external muscles of the leg. Rigidity at the knee is less objectionable. Every additional week of suppleness may mean increased voluntary control. Now, also, the patient should be constantly encouraged to concentrate his attention upon the paralyzed side and repeatedly attempt to move the paralyzed muscles, aiding the effort by the simultaneous use of the sound side in executing the wished-for movements. Weakness of the sound side may defeat efforts at walking, unless the patient is greatly encouraged and strongly supported. The distrust of their strength and preference for inactivity must sometimes be actively met by the encouragement and authority of the physician. After two years no further gain is to be expected, but even then laceration may be facilitated by orthopedic apparatus or by a tenotomy of the heel-tendon. There are very few hemiplegics who escape more or less persistent modulation with *colic*. Aside from *cephalalgia* cases it can not be strongly urged. As a treatment of the basic arterial state,

however, it often has a legitimate place, and therein may be prophylactic of a second attack of hemorrhage. Too often it seems to degrade the patient's general health without corresponding benefit. It should go without saying that general hygienic and tonic measures are always indicated. The enforced inactivity of the hemiplegic favors intestinal atony and cutaneous sluggishness, which require watchful and intelligent management.

## CHAPTER VII.

### CEREBRAL SOFTENING.

THE term "cerebral softening" is open to objection for several reasons, but usage has confirmed its rank. "*Softening of the brain*" is a lay expression usually applied to dementia, and based upon somewhat erroneous notions of the condition of the brain in such cases. By *cerebral softening* is here meant the retrograde process in vascular territories of the brain, occasioned by arterial obliteration and local deprivation of blood-supply. It is an infarction process exactly similar to that which is common in the spleen and kidney. The brain-structure, however is not of a uniform functional value, and a cerebral infarct in the majority of instances destroys specific brain-powers instead of simply reducing the capacity of the whole, as in the abdominal organs mentioned. In addition it gives rise to secondary conditions similar to those following cerebral hemorrhage. It is to Virchow that we owe our first exact knowledge of the genesis of cerebral softening. Previously the softened atrophic area was attributed to inflammation or some vague morbid process. We know now that the immediate cause of the *encephalomalacia* is the occlusion of the cerebral vessel that supplied the diseased portion. This obliteration of the lumen of the artery arises (1) from thrombotic obstruction developing on the site, or (2) from plugging by an embolus starting at a distance, or (3) from local thrombosis following the lodgment of irritant emboli.

When a cerebral artery is occluded, the blood-supply of a tolerably definite territory is cut off because of the anastomotic defects of the cerebral circulation. In the case of a sizable vessel the center of its arterial field is wholly deprived of blood, but the margin is only relatively impoverished, as there is some overlapping of these vascular territories, by slight anastomoses and in some cases by direct anastomotic relations with the veins. The moment an artery is closed its distal portion is deprived not only of blood, but of blood-pressure, and, aided by its own resiliency, it tends to collapse. This gives opportunity for back pressure from the return or venous circulation, and the territory deprived of arterial inflow may become the seat of venous congestion. Moreover, the arterioles deprived of their nutrient contents are disposed to promptly degenerate, and thus arise the punctate hemorrhages so commonly found in comparatively recent cases of cerebral thrombosis. Subsequently, retrogressive changes take place looking to the removal of the mortifying focus. Fatty degeneration and phagocytosis go on rapidly.



The coloring-matter of the extravasated blood finally alone remains. Repair takes place about the focus of disease, and it becomes encapsulated. In some infarcts of minor size cicatrization ensues, and a scar is left to mark the location of the vascular lesion.

**Pathological Anatomy.**—The first effect of arterial occlusion is to cause anemia in the distal portion, and the territory of distribution becomes blanched. The tissues degenerate, necrosis follows, and *white softening* is produced. If, from the return circulation and local punctate hemorrhages the strangled area becomes suffused and infiltrated with blood, color is added and *red softening* is presented. Finally, as resorption takes place and degeneration becomes complete a yellow color from the remnants of the hemo pigment marks the softened and perhaps encapsulated tissue, giving rise to the name *yellow softening*. The three appearances are but stages of the same process, but *white softening* is not necessarily followed by the red and yellow changes. The red appearance, due solely to the hemorrhagic element, appears, if it appears at all, after a few hours or a few days, depending upon the activity of the venous back pressure and the degeneration of the arterioles.

In a very few days after arterial obstruction has occurred the corresponding cerebral tissue presents a marked appearance of degeneration. It is a fact of practical bearing that nerve-cells, deprived of their nutritive supply for a few hours, are permanently ruined. The softening focus is infiltrated with serosity and the cellular and neuroglial elements are already breaking up. The myelin separates into droplets and is rapidly taken up by migratory leukocytes, which attain large proportions and have by some been described as granular bodies. The degenerating focus shrinks and softens. If situated beneath the pia, the resulting depression is filled with a turbid, milky fluid, and the soft meninges are thickened and highly vascular. It becomes impossible to strip the pia from the gyri of the softened area without decorticating them. The cortex is pale and friable, the white substance softened often to diffidence. In old foci of softening the surrounding tissue is thickened, especially in its neuroglial makeup, and presents an indurated wall within which a yellowish fluid substance containing fat-crystals and amorphous matter represents the former cerebral structure. Yellow softening may be found in the end of six weeks, but is a feature of old lesions. These may even become calcareous. *Cicatrization*—the fibrous obliteration of small softenings—has been already mentioned. In some cases the softened focus becomes infected, as by an embolus from infectious endocarditis, or from pneumonia, or from any infection atrium, and a secondary true *encephalitis* is developed. This rapidly goes on to *abscess* formation, frequently with putrid, offensive, gangrenous contents. Thrombosis occurring in the perforating arteries of the base, which are entirely without anastomoses, or in the deep cortical branches which penetrate the centrum ovale, produces rounded islets of necrosis, which in time may become encapsulated and contain only serous fluid.

At the seat of arterial obliteration arising from local thrombosis we find, in recent cases, a partially or completely organized clot adherent to a spot or ring of endarteritis or atheromatous thickening. An embolus

does not at first present adhesions to the intima. It may be made up of pure fibrin from the endocardium, or calcareous particles originating in cardiac or arterial atheroma. Hydatids, filaria, and any minute substance finding access to the blood-stream may furnish a cerebral embolus. In about nine cases out of ten the embolus originates in or near the heart, and in fortunate cases the exact location of its origin has been detected. Ordinarily an embolus lodges at the branching of an artery, from which point the thrombotic fibrin deposit extends. If the embolic mass be calcareous, it may abrade the intima and give rise to a dissecting aneurysm, which in turn leads to occlusion and thrombosis. The size of the softening depends entirely upon the size and relations of the occluded vessel.

The location of cerebral softening is more frequent on the left than on the right side of the brain. The left middle cerebral is especially selected, and its perforating or basilar are more frequently involved than its cortical branches. This is due perhaps to the fact that cardiac emboli following the direct route tend to enter the vessels that most conform to the straight line of their momentum. These are also the favorite seats of atheroma. The anterior capsuloganglionic region is the most common site of cerebral softening, followed in decreasing frequency by the posterior capsuloganglionic region, the cortical territory of the middle cerebral, that of the posterior cerebral, then that of the anterior cerebral. Softening is rare in the cerebellum and in the bulb, except in syphilitic endarteritis, which frequently implicates the brain-stem. In rare cases we encounter a bilateral and symmetrical softening. In some instances this may be explained by thrombosis or embolism starting in the circle of Willis. For instance, an atheromatous patch at the bifurcation of the basilar may furnish a plug to the left side, and circulation on that side being proportionately reduced, the next embolus goes to the right side. In some cases there are numerous foci of softening. Softening may affect only the gray cortex, or the white subcortical substance, or both. The extent of necrosis depends always on the damaged arterial supply. If the Sylvian trunk be occluded, the entire sensorimotor zone is softened as well as the subcortical white substance as deep down as the basal ganglia. If the thrombosis or arterial stagnation implicates only the short cortical branches, local death of the brain-matter alone may follow. Again, if the arterial disturbance affects the long penetrating arteries that traverse the cortex to supply the central ovule, the cortex may be spared.

**Etiology.**—The ultimate causes of cerebral softening are those of the arterial diseases, already considered, that underlie thrombosis and embolism. In a rough way we may say that thrombosis, namely a sequence of atheroma and arteriosclerosis, pertains to advanced age, alcohol, lead, gout, and syphilis, and that embolism is an accident of left endocarditis due to acute rheumatism and the infections of pneumonia, typhoid, diphtheria, the puerperium, etc. As a presumptive rule, we may also say that cortical softenings are commonly due to embolism and occur most frequently in youth; that those of the central area are due to thrombosis and take place in advanced years. Thrombosis is favored by a weak heart and sluggish arterial current of low



tension, conditions ordinarily found in cachectic and marantic patients and favored by sleep. Embolism is favored by any act that throws a load upon the heart and produces cardiac stimulation. Vigorous muscular efforts, sudden emotion, or merely rising to the feet from a reclining position may start an embolus when friable arterial or cardiac vegetations exist. Very exceptionally a cerebral artery is obliterated by conditions arising outside its lumen, as by a rapidly growing tumor or other compression factor acting with some degree of promptness. Unless the compression is brought rapidly to bear, the circulation is able to adjust itself. Heredity, sex, age, temperature, barometric pressure, and the seasons are of much less significance than in cerebral hemorrhage.

**Symptoms.**—The early symptoms of cerebral softening are dominated by the rapidity with which the arterial current is blocked and by the importance and size of the vessel involved. As a matter of fact, embolism is capable of producing instantaneous blocking of the arterial lumen, and symptoms of great violence at the onset to some degree indicate embolism if hemorrhage is excluded. On the other hand, while thrombosis usually produces progressive symptoms, a slowly growing thrombotic arterial lining may cause a sudden deposit of fibrin from the blood, and intense symptoms are thereby precipitated. Again, a small embolus may only partially choke the blood-current, and the slowly developing occlusion is marked by the deliberately advancing symptoms and prodromata of the stroke. Cerebral thrombosis, therefore, presents both (1) an abrupt and (2) a progressive onset, with different cerebral symptoms immediately resulting, but with the same terminal conditions.

**The Abrupt Onset.**—With none or only momentary confusion the patient has a stroke or cerebral attack, followed by loss of consciousness, perhaps even by the development of coma. Hemiplegia develops, consciousness returns, and more or less paralysis persists. In about an equal number of cases consciousness is not lost, though the paralysis is as quickly developed, and, as a rule, the mental subjugation, the cerebral insult, is less than in hemorrhage. Many cases are attacked during sleep, and awake at their usual hour with no appearance of stupor and with their ordinary mental brightness. A very significant feature of thrombosis affecting the cortical circulation is the Jacksonian or limited convulsions which are commonly presented, and which tend to repetition during the early hours and days of the illness without great, often without any, disturbance of consciousness. Distinguishing softening from hemorrhage, we seldom encounter the retarded strong pulse, the subnormal temperature, the unilateral heat, the congested face, the respiratory difficulty, and the prolonged coma so characteristic of a ruptured cerebral vessel. In a fair proportion of all cases of thrombosis the early symptoms are progressive. A monoplegia becomes a hemiplegia. Paralysis beginning in the leg invades the entire extremity, reaches the upper limb, implicates the face, disturbs speech if on the left side of the brain, and finally the hemiplegia is complete. This is due to the thrombus, by gradual increase, backing down the artery from its original place of development, and if it extend into the circle of Willis

the opposite Sylvian artery may also be blocked. When the basilar is invaded bulbar symptoms usher in a fatal termination. Thrombosis beginning in the branches of the middle cerebral may first produce an aphasia; lower facial paralysis and loss of power in the limbs of the same side may then be gradually added. The distribution of the arteries to the motor region makes clear the sequence of events. (See page 198.)

**The Progressive Onset.**—When the arterial obliteration is a slow process and the caliber of the vessel is gradually reduced, disturbances in the poorly irrigated cerebral district are likely to be manifested. In the case of a Sylvian vessel this is the rule. The patient complains of more or less transient or persistent sensations of fullness, heaviness, formication, weakness, pain, or other vague discomfort in the face or in one extremity, or merely in the distal part of an extremity. The face at the same time may show transient weakness, the leg may be dragged a trifle, and a little clumsiness may be noticed in the finer finger-movements. Speech very often is disturbed, presenting various slight aphasic indications. Any or all of these symptoms are likely to be most marked toward night or after fatigue. They indicate a local anemia of their respective cerebral mechanisms and a lowered nutrition that translates itself in paresthesias and paresis and foreshadows paralysis. In many cases the cortical irritation shows itself in twinklins in the face or in the extremities, or in a sudden thickening of speech.

Groups of such symptoms may appear several times, lasting at intervals for a day or two and then recede for weeks or months. Finally they return with more hearkness and severity, convulsions may ensue, the thrombosis is complete, and paralysis is permanently established.

This progressive loss of cortical circulation is to some extent a part of the physiology of senile involution. The blood-current becomes gradually weaker and the arterial channels progressively diminish in capacity. The heart loses its force; the fine, delicate, cortical vessels are most affected; circulatory stagnation in the brain-mantle is favored, nutrition fails, psychical and motor and sensory functions are reduced in activity, and dementia is inevitable. In the brains of the aged, multiple foci of softening are commonly encountered.

The paralytic state may thus be established suddenly with or without an apoplectic state, or may advance by steps and be preceded by numerous warnings and significant symptoms. Softening once established has the tendency, as has already been indicated, to invade adjoining cerebral areas, related through the arterial supply. Hemiplegia and the hemiplegic state are common results. In cases of sudden onset, especially if marked by an apoplectic seizure, the hemiplegia is usually complete and severe. In cases of less active onset, and especially in those of gradual development, the functional loss is less profound and there is a greater tendency to subsequent recession, leaving only a monoplegia or a monoparesis. The paralysis may be monoplegic from the first, and softening is the most common source of the cerebral monoplegias. In the same way aphasia alone may indicate



the cortical disease. Persistent aphasia in all its varieties, nine times out of ten, is due to cerebral softening. In the same way the cortical areas of half-vision may be destroyed, or any given cortical function may be singly selected for abolition.

The paralytic state due to softening presents practically the same course and final deformities that mark it when resulting from cerebral hemorrhage (see p. 213). At first flaccid, the permanently paralyzed limbs show increased reflexes after about three weeks, and the usual contractures are developed. If the paralysis in a given member remains unimpaired at the end of the first week, it is likely to be permanent. The tendency to recession, to progressive improvement, is by no means so marked as in hemorrhage. While the traumatic shock to the brain is less, the destruction is relatively greater.

**Sensory disturbances**, which in hemorrhage ordinarily disappear promptly, are commonly persistent in the paralytic state after softening. Their persistence indicates a cortical lesion unless hemiplegia and hemi-anesthesia coincide, when the sensory crossover is involved, whatever be the lesion, hysteria being excluded. The sensory disturbances, after cortical softening, are paralytic, not anesthetic. This is due to the stratification of cortical function, sensation, muscular sense, and motion being represented at increasing depths in the sensorimotor zone, as described on page 116. That sensation is not abolished, but is only disturbed, is due to its probably complete bilateral representation in the hemispheres.

**Course.**—As cerebral softening is a brain accident, the result of arterial disease, like hemorrhage, it may present all gradations of severity and extent and numerous clinical forms. Cases marked by abrupt onset with a well-marked apoplectic phase may sink into coma and die. In proportion as the onset is gradual the tendency to immediate death is lessened, excepting in those instances where progressive invasion of vascular territories shows that the thrombotic process is advancing toward the circle of Willis. The paralysis or aphasia once definitely developed, there is still a tendency to improvement, which is less marked than in corresponding cases of cerebral hemorrhage. Should the softened focus become infected, an acute purulent encephalitis is likely to carry off the patient. This complication is marked by elevation of temperature, sometimes by somnolence, usually by convulsions referable to irritation in or about the diseased area, and is frequently associated with a pneumonia or an acute bedsores. Months and years after the onset of the disease the softened focus still constitutes an irritant brain-lesion which may cause epileptoid convulsions. It may have a bad influence on the integrity of the mind and be followed by insanity. The hemiplegic state, when once developed, presents the common features and indications described in the preceding chapter. When multiple softenings occur, the clinical picture is much modified. A right facial paresis, with aphasic symptoms, may accompany a left brachial monoplegia.

**Diagnosis.**—The diagnosis of cerebral softening often presents many difficulties and sometimes is impossible. In the great majority of

cases, however, a careful scrutiny of all the data enables us to make a positive diagnosis, and in a fair share of the remaining cases a strong presumption can be established. The question primarily concerns the arterial occlusion. After the age of ten and up to forty a paralytic attack suggests embolism or syphilis. Endocardial disease, a recent history of acute rheumatism or infectious fever, speaks strongly for embolism. Practically a diagnosis of embolism can not be made in the absence of cardiac symptoms. After the age of seventy years the presumption is again in favor of softening, but from forty to sixty-five or seventy hemorrhage is the commoner cause of paralysis. A history of syphilis, plumbism, or alcoholism suggests softening.

The mode of onset may clearly indicate softening. Prodromata and the gradual paralytic invasion, localized spasms, monoplegias, aphasia, and limited parasthetic areas are indicative of softening. The disproportion between the paralysis and the apoplectic features suggests softening. A complete hemiplegia of rapid development without a stroke can hardly be due to hemorrhage. A pale face, weak heart, normal temperature, and practically undisturbed consciousness all favor softening as the cause of a paralytic attack. Multiple palsies and bilateral palsies, especially if symmetrical, are usually due to softening. Softening occurs often during sleep and under conditions of low arterial tension, except in embolism. After the paralysis has been established it is likely to persist, though in children and young adults it may notably improve.

Many of the clinical features of softening are produced by tumors, but ordinarily new growths have a more insidious course and present the cardinal symptoms of headache, vomiting, optic neuritis, and vertigo. Cerebral abscesses usually furnish a history or evidence of injury or stasis, but a softened area may be infected and break down into an abscess. It is with cerebral hemorrhage that softening is most confused. They have many common points. For instance, syphilis and alcoholism may cause both, and both have apoplectic onsets and paralytic sequels. In some instances the differential diagnosis can only be made out in the light of the subsequent course of the disease, and in a small number of cases it seems quite impossible to make it. The following table of probabilities will serve to show the direction a presumptive diagnosis should take:

TABLE OF DIFFERENTIAL PROBABILITIES IN CEREBRAL HEMORRHAGE AND THROMBOSIS.

	HEMORRHAGE.	THROMBOSIS.
FAVORING FACTORS	Frequent before three years of age, and between forty and sixty.	Common in old age and in young adults.
	Peri-arteritis and urinary abscess give the usual antecedent.	Endocarditis, atheroma, embolism, and cachexia.
	Insidious often marked.	Insidious rare.



TABLE OF DIFFERENTIAL PROBABILITIES IN CEREBRAL HEMORRHAGE AND THROMBOSIS.—(Continued.)

	HEMORRHAGE.	THROMBOSIS.
INITIAL CONDITIONS.	High arterial tension.	Low arterial tension.
	Excitement, effort, or shock.	Barely excitement or effort, except in embolism. Sleep common.
ONSET CONDITIONS.	No prodromata.	Prodromata common.
	Sudden stroke fatal.	Complete stroke rare.
	Coma marked.	Coma slight or wanting.
	Rectal temperature reduced, and surface temperature elevated on the paralyzed side.	Temperature usually unchanged.
	Congested face; respiratory difficulties.	Pale face; no respiratory disturbance.
	Pulse slow, full, bounding.	Pulse weak, soft, often rapid.
	Motor loss usually hemiplegic and fully developed at once.	Motor loss often monoplegic and inclined to extend.
	General convulsions common.	Localized convulsions common.
	Spinal fluid often bloody.	Spinal fluid clear.
OUTCOME.	Rapid improvement in motion.	Slow motor improvement. Extension of paralysis often observed.
	Foot usually gains tone rapidly than hand.	Foot often gains less than hand.
	Anesthesia usually fleeting.	Parosmia periods.
	Persistent aphasia exceptional.	Persistent aphasia and other cerebral symptoms common.
	Postplegic atonics, trembling, and chorea common.	Postplegic atonics, trembling, and chorea uncommon.
	Postplegic convulsions rare.	Postplegic convulsions common.
	Spasmodic weeping and laughter common.	Spasmodic weeping and laughter exceptional.

**Prognosis.**—Cerebral softening is an accident following such a wide variety of diseases and conditions which provoke the endarterial process of thrombosis that prognosis can not be generalized. Every case has its own indications. The tendency to immediate death is less than in hemorrhage, but the appearance of pneumonia, or an acute lesion, or a sudden elevation of temperature, even of moderate degree, indicates a grave complication and a probable fatality. In embolic cases, if it is probable that the embolus is infected, as in infectious endocarditis, diphtheria, and the exanthemata, the outlook is much darkened by the probability of acute infectious encephalitis being set up in the softened area, to be followed by abscess and probably by death. Advanced

years are against the patient. In every case the prognosis should be held in reservation for a week until it is evident that the thrombosis is not spreading and that local infection has not occurred. The temperature is here a valuable guide. Persistent severe convulsions commencing early, perhaps present at the onset, are of grave significance. They point to involvement of the cortex and meninges on the one hand, or of the lateral ventricles on the other. The secondary implication of the meninges or ependyma over the softened area is usually limited, but in infected cases it may lead to a generalized inflammatory process of the utmost gravity. When the first fortnight has passed, the paralytic state may be considered established. Contractures and deformities are thereafter developed, as in hemorrhage. The hemiplegic state presents nothing dissimilar to that following arterial rupture, and has been described in a previous chapter. The condition presented by a given case of softening at the end of the first month is likely to be permanent. This is especially true after middle life. There is also the possibility of epileptoid attacks following at any time, and the persistence of the endarterial disease or its generalized presence constitutes a continual menace. This is particularly true in multiple softenings and in the bilateral forms such as that which furnishes a pseudobulbar palsy.

**Treatment.**—The treatment of cerebral softening to be efficient must antedate the occurrence of thrombosis. In a word, it must be prophylactic. In another word, it must be the treatment of the arterial disease. When the arterial current is cut off we yet have to deal with the local disease in order to prevent an increase of the thrombus or its repetition and to cause, if possible, its diminution. When called at the onset of the softening in the early hours of the attack, if hemorrhage can be excluded, the treatment consists of maintaining a masterly inactivity. The patient's position should be horizontal, to favor the cerebral circulation; the flagging heart may be encouraged with strychnin and relieved with the nitrites given frequently, which also favor increased circulation in the ex-anguinated brain territory. The repeated use of normal salt solution by the bowel, or better by the hypodermic method, is often of great value. Small quantities of ascaridment should be administered, and the functions of the bowels and bladder supervised. If hemorrhage cannot be excluded, the same course is still advisable, but if hemorrhage is diagnosed, the opposite plan of treatment for that condition, already described, should be instituted. Purgation and vesicsection cannot benefit a cerebral territory already ex-anguinated. In cases of embolism, cardiac repose is to be encouraged that other particles may not emigrate. Bromid, to control the convulsions, may often be required. The further management of the case is that of good nursing. The arterial state must never escape attention. Its amenability to treatment governs the outlook for the patient and the probability of recurrence. When the paralytic state is established, its management is the same as that laid down in the previous chapter, and for the terminal monoplegia or hemiplegia the indications are likewise identical. The treatment of aphasia and the development of the opposite-sided speech-centers have been described in the chapter on Aphasia.



## CHAPTER VIII.

## DISEASES OF THE CEREBRAL VEINS AND SINUSES.

**Anatomical Considerations.**—The blood entering the cranium by the internal carotids and vertebrals after irrigating the encephalon makes its exit mainly by the internal jugular veins. The return circulation from the ventricular portion of the cerebrum and the callosal portion of the hemispheres is by the Galenic veins and inferior longitudinal sinus, all of which empty into the straight sinus. From the convexity the pial veins run upward and open into the superior longitudinal sinus.

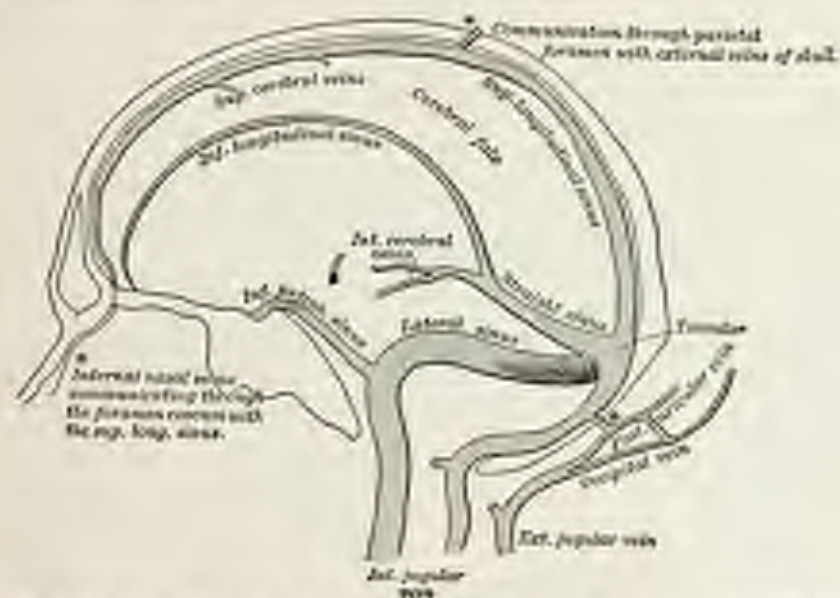


Fig. 55.—Diagram showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by \* (Lamb).

in a forward direction against the slow blood-current of this dural channel. Here the circulatory conditions are rendered still more unfavorable by hydrostatic pressure, by the presence of trabecule in the sinuses which impede the flow of blood, and by venous retardation during inspiration. The cerebellar veins empty mainly into the lateral sinuses. Into the dural sinuses also empty many veins from the face and scalp. The facial vein communicates with the cavernous sinus through the ophthalmic vein. The veins of the nasal vault open into the anterior extremity of the superior longitudinal sinus. Numerous veins of the scalp along the median line have a similar outlet. Veins from the mastoid process and its cutaneous surface enter the lateral and

petrosal sinuses, and the occipital and posterior auricular veins are connected with the lateral sinus. In addition, many veins of the cranial diploë discharge into the sinuses. Finally, the sinuses connect with the veins of the spinal canal.

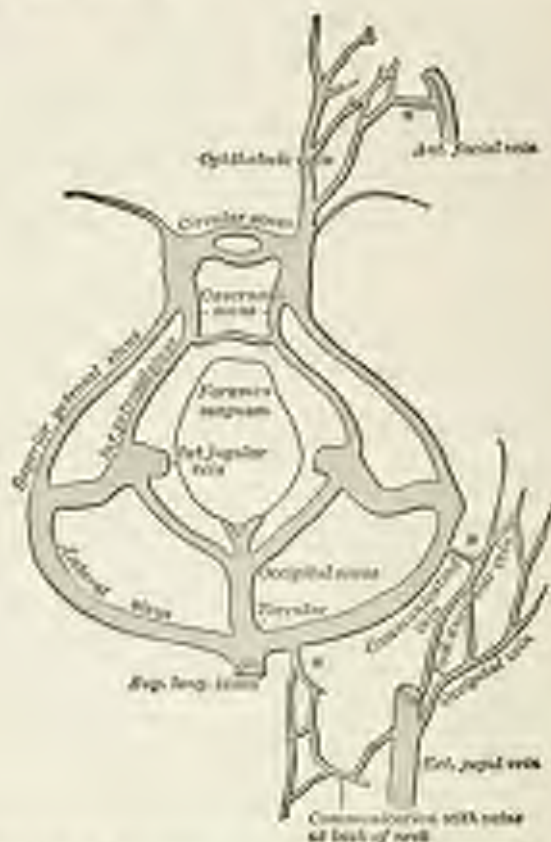


Fig. 48.—Diagram showing the communication existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by \* (Linder).

The cerebral veins are subject to the same lesions that influence veins elsewhere, but we are only called upon to consider phlebitis and venous thrombosis. They are usually associated and, practically speaking, severe cerebral symptoms are alone produced by thrombosis. Thrombosis may occur in the pial vessels of the convexity and extend into the longitudinal sinus, or, beginning in the sinus, may invade the cortex and give rise to localized softening and focal manifestations. The entire subject may be conveniently described under the head of Sinus Thrombosis.

#### SINUS THROMBOSIS.

Intramural sinus thrombosis occurs more frequently even than thrombosis in the pelvic veins, or in those of the lower extremities. It



is favored by the sluggishness of the venous current and the other anatomical peculiarities above indicated. Septic invasion is also extremely liable from the relation of these channels to the cavities of the nose, throat, and ear, and to the frequently traumatized surface of the face, neck, and scalp. It occurs in two forms,—the marantic and the infective.

**Marantic sinus thrombosis**, or *primary thrombosis*, is a local condition occurring usually in the superior longitudinal, rarely in the lateral, and very rarely in the occipital sinus. It occurs in debilitated states, and is most common at the two extremes of life. Exhausting diseases, weakness of the heart, and in general any cachectic state predispose to it. Prolonged illness, as from diarrhea, typhoid fever, pneumonia, phthisis, cancer, malaria, the anemias, etc., often precede it. Under these conditions there is a tendency to fibrin deposit and thrombus formation which, once started, is likely to extend. Should it commence in a cortical vein, or extend from a sinus to the brain-surface, Jacksonian fits may be produced.

Usually on post-mortem examination the thrombus is found to involve several sinuses and their tributary veins. When the entire lumen of a cortical vein is blocked, the drainage of its territory is prevented and localized edema, punctate hemorrhage, and red softening follow. In the same way edema occurs in the superficial parts of the face and head which drain into an occluded sinus. Swelling about the eye and exophthalmos, with retinal thrombosis and epistaxis; swelling about the mastoid, over the vertex or occiput, is produced by the thrombotic closing of the sinuses respectively related to these regions. When the lateral sinus is involved, the thrombus may extend down the internal jugular and be found as a firm, polypoid mass in the neck. In marantic thrombosis the clots are firm and non-adherent to the walls of the vein or sinus,—that is, they are not inflammatory. They tend to organize or resorb and do not disintegrate. Reestablishment of the circulation in the sinus is therefore possible, and usually takes place in long-standing cases, but in cortical veins, if cerebral softening occurs, there is no tendency to circulatory restoration.

The symptoms of marantic thrombosis are those of venous stasis, localized edema, and disturbed brain-function following upon exhausting diseases. As the longitudinal sinus is usually affected there is often epistaxis and disturbance in the leg-centers, indicated by weakness, tremors, paralysis, and spasms in the lower extremities. Convulsions in children are commonly encountered, and may be limited or monoplegic when a cortical vein is invaded. Meningeal irritation often shows itself in rigidities, retracted neck, and vasomotor disturbances.

The diagnosis of marantic thrombosis is usually obscured by the overshadowing picture of the preceding illness, and in those cases where external edema does not point the way it is rarely deciphered during life. There can be little doubt that it furnishes some of the cases of cerebral palsy in childhood. Convulsions coming on late in the history of exhausting illness, especially in children, should direct attention to

the possibility of sinus occlusion. If meningeal or focal symptoms are present, coupled with local edema about the eye or face, over the vertex, in the mastoid or occipital region, a diagnosis may be made.

The prognosis will depend upon the nature of the initial illness, the probability of cortical softening, and the vital prospects. When the carlexia or exhausting disease can be controlled and life maintained, the tendency to resorption of the clot presents a favorable outlook, except for the softened areas of brain-tissue. When these have been produced, permanent disability of a motor or mental character, or both, may be expected. The treatment is that of the general condition.

**Infective sinus thrombosis, inflammatory thrombosis, or secondary thrombosis,** is the result of the invasion of pathogenic infectious organisms. It is, therefore, a secondary process and occurs usually in adults. Generally it is located in one of the paired sinuses and in the one nearest the infection atrium. It is likely to produce meningitis or give rise to cerebral abscess, or both, and it is frequently attended or followed by systemic infection and pyæmia. It arises from septic traumatic conditions of the face, scalp, cranial and facial bones, and from septic processes in the nose, mouth, pharynx, in the various lony sinuses, and in the middle ear and mastoid process. By far the most common source of infection is suppurative middle-ear disease. Anthrax of the face and lips, facial and scalp erysipelas, dental caries, and carbuncles have been noted as causes. The sinus always becomes infected by extension of the septic process to it, either by direct incursion or by propagation along a venous tributary. Inflammation of the sinus-walls is followed by thrombotic coagulation of the contained blood, and a septic plug is formed that closely adheres to the diseased and softened vessel.

The septic, soft, and disintegrating thrombotic clot, swarming with pathogenic and pyogenic bacteria, sets up infection of the immediate territory, and, yielding particles to the blood-stream, often induces disseminated and systemic infection. Thus arise the numerous pyogenic foci throughout the body that are frequently present. The thrombus once formed is prone to extend, and the internal jugular is often invaded, presenting itself in its upper third as an indurated cord that can be palpated in the neck. Even the superior vena cava has been invaded. The sinus-walls being softened, the neighboring leptomeninges are infected and a localized or disseminated septic meningitis is added. In a similar way the infective process travels along the veins into the substance of the cerebrum and sets up abscesses. Macoswen thus tabulates the principal differences between mamule and infective sinus thrombosis:

#### SINUS THROMBOSIS.

##### MAMULE.

1. Chiefly affects the longitudinal sinuses.
2. The clots tend to organization or are absorbed.
3. Hemorrhages into cerebral cortex in about half the cases.

##### INFECTIVE.

1. Chiefly affects the paired sinuses.
2. Clots tend to purulent disintegration.
3. Hemorrhages into brain or cerebellum seldom occur.



- |   |  |
|---|--|
| 4. Tendency to produce brain softening.                             | 4. No tendency to brain softening.   |
| 5. There is seldom purulent infection as a sequence.                | 5. Purulent infection common from septic or infective enditis.                 |
| 6. No accompanying leptomeningitis, cerebral or cerebellar abscess. | 6. Often co-existent purulent leptomeningitis, cerebral or cerebellar abscess. |

The symptoms of infective sinus thrombosis are local and systemic. The local ones are due to circulatory disturbance, such as circumscribed edema and brain symptoms. They will be given in detail in the description of thrombosis of special sinuses. The systemic symptoms are those of septicæmia; intense headache, often localized at the seat of disease; vomiting, fluctuating and remittent temperature, small, thready pulse, leucocytosis, rigors, profuse perspiration, dry tongue, anorexia and diarrhea, or constipation.

Depending upon the preponderance of symptoms and their grouping, the septicæmia shows different clinical varieties. When the lungs are first or mainly involved by the plugging of pulmonary vessels, localized or diffuse pain is occasioned, cough is induced, and the expectoration may change to "prune-juice" appearance, and then becomes purulent, fetid, and extremely offensive as the pulmonary process increases. Abscesses form and gangrene occurs. In this way septic pneumonia also is induced.

When the brunt of the attack falls upon the abdominal organs, the typhoid type of septicæmia, which closely mimics enteric fever and is sometimes mistaken for it, is presented. In another and much smaller group of cases meningeal symptoms dominate the picture and are actually due, in large part at least, to the infective leptomeningitis. All three of the symptom groups, or any combination of their various features, may be presented by the same case.

Infective cavernous sinus thrombosis arises from septic invasion, reaching the sinus usually by way of the ophthalmic vein. It may also be due to a forward extension of a septic process in the lateral or petrosal sinuses. The secondary meningitis to which it gives rise is focal. Fractures of the cranial base and blows on the head have furnished its starting-point. Erysipelas of the face, especially about the eye and nose; abscess of the orbit; infections of the nasal, buccal, and pharyngeal cavities or of their sinuses; ulceration of the tonsillar glands, and caries or periostitis of the facial, especially of the maxillary bones, have led to it.

Symptoms: One sinus is usually first involved, and the local manifestations are one-sided. There is, however, a pronounced tendency for the process to invade the opposite sinus, and then the case presents bilateral signs. Such a sequence is highly diagnostic. The first affected side may even show improvement through the establishment of the collateral return circulation before the second side is invaded. There is usually considerable pain of a supra- or infra-orbital neuralgic sort, and diffuse headache. Mental symptoms are wanting, unless meningitis is set up, and then delirium, lethargy, and coma may appear. When the sinus is plugged the return circulation through the

ophthalmic veins is cut off. The orbital contents become edematous, the ocular globe is thrust forward, the lids are swollen, and the swelling extends to the nose, brow, and cheek. There may also be swelling on the same side of the pharynx. The optic disc is congested or choked, the retinal veins are distended, and pressure is exerted on the ocular nerves that enter the orbital apex. This causes more or less *ophthalmoplegia*. The third, fourth, sixth, the ophthalmic division of the fifth, and the optic nerves are more or less affected. Ptosis, strabismus, pupillary stasis, and defective vision in varying degrees are thereby added to the exophthalmos.

The invasion may be abrupt or insidious and the disease may last from a few days to several months, but infective cases are practically fatal. When the second eye is involved, it usually is very rapidly affected. The appearance of basilar meningitis and the development of septicaemia add immediate gravity to the already serious condition.

**Infective lateral sinus thrombosis** is the form most frequently encountered. Its origin is nearly always in a septic condition of the middle ear. The petrosal sinuses and the internal jugular are usually invaded. It is commonly encountered in young adults, and is rare in the two extremes of age. While ordinarily due to middle-ear disease, it may arise from a mouth or throat infection by way of the Eustachian tube and tympanum, from extension of thrombosis in other sinuses, from basilar fractures involving the petron, and from infections about the occiput, nucha, and mastoid.

**Symptoms:** The lateral sinus is usually affected from a chronic middle-ear suppuration which has caused more or less erosion of the tympanic bony structure. An acute process may cause it, but this is exceptional. In the chronic cases of purulent otitis media it is a constant menace. Frequently there is a lessening or cessation of the ear-discharge, pain develops in the ear, and headache follows. The fluctuating temperature mounts, and vomiting and rigors indicate the involvement of the sinus or an intracranial extension. Local signs of lateral sinus thrombosis depend on the obstruction of its lumen and the location or extent of the thrombus and phlebitis. Occlusion of the sinus, blocking the inlet of the mastoid vein, gives rise to a circumscribed edema extending from the auricle over the mastoid. Pain on percussion of the mastoid is present only when the bone or periosteum is inflamed. If the thrombosis is situated lower in the sinus and shuts off the condylar veins, through which the superficial circulation of the lateral lower occipital region drains, a brawny hardness and edema may be made out in the upper part of the posterior cervical triangle. Should the phlebitis extend into the internal jugular, extreme local tenderness is found over the upper portion of this vessel on palpation, or may be experienced by the patient in swallowing. The head is usually inclined to the affected side to lessen muscular pressure on the jugular. When the thrombosis follows down into the jugular, it may be easily palpated as a firm, cord-like structure. Disintegration of the thrombus may cause it to disappear in a few days. The lymphatic glands in the neck are frequently enlarged and easily palpable. Gerhardt has pointed out that during inspiration the external jugular vein on the affected side is



less prominent. This is due to the occlusion of the internal jugular, which allows rapid drainage of the external branch into the common trunk. If, however, the thrombus extends into the common trunk, the external vessel is then engorged and more prominent than on the sound side.

If the inflammatory thickening at the jugular foramen is sufficiently great, it involves, by extension or pressure, the cranial-nerve trunks, which make their exit at this opening. Pnucnogastric, spinal accessory, and glossopharyngeal symptoms are then produced. Respiratory, laryngeal, cardiac, and vocal disturbances; difficulty in swallowing, quærs or paresis in the sternomastoid and trapezius point to this condition. Abscesses sometimes form in the neck, under the sternomastoid or in the medial region.

Almost from the first there are distinct cerebral symptoms. The cephalalgia is attended by somnolence, which may deepen into coma. Delirium is often an early symptom. Phlebitis is likely to extend into the temporoparietal lobe and cerebellum, giving rise to diffuse inflammation or rapidly producing softening and abscesses. At the seat of the sinus thrombosis the softened dural wall no longer protects the soft meninges, and a localized septic meningitis, with a tendency to become diffuse is occasional. Even the bone under the sinus is eroded, and definite discolorations are left, both on the cranial wall and on the cerebral surface, to mark the site of the sinus disease.

A disease presenting so many complications and possibilities necessarily lacks clinical uniformity. While lateral sinus thrombosis is usually confined to one side, it may propagate itself into the venous channels of the opposite side and infect both internal jugulars. The appearance of cerebritis, meningitis, or intracranial abscess greatly reduces the life chances. Septicæmia is particularly likely to develop, with its own serious import. The violence of the infection, however, seems to vary between wide extremes. Some cases run a rapid course to fatal termination in four to seven days, others last weeks and months, and exceptional ones may recover. The gravity of the disease can not well be overestimated.

Infective thrombosis of the longitudinal sinuses is extremely rare, while marantic thrombosis finds its most common location in these venous passages and especially in the superior one. Infectious conditions in the nasal vault, in the scalp, and in the cranial diploë of the vertical region are capable of extension to the superior longitudinal sinus. Phlebitis may then extend to the cortical veins. Occlusion of the sinus leads to local edema in the scalp and probably in the brain also, but the collateral circulation obviates any serious results from this mechanical feature. The danger lies in the likelihood of septic cerebral phlebitis and septic meningitis. Usually the obstructive meningitic features predominate, and suggestions of cortical phlebitis and even of abscess formation are obscured or overlooked.

The treatment of infective cranial sinus thrombosis is primarily surgical. The infection atrium is to be determined and rendered thoroughly aseptic. Whenever possible, it must be eradicated. In the case of the cavernous sinuses little more can be done by the surgeon,

but Dwight and Germain<sup>1</sup> have reported one instance in which the cavernous sinus was treated surgically with immediate improvement in the patient's condition, though with fatal termination ultimately. A previous case by Hartly was entirely successful. Aggressive interference is allowable, and indicated when the lateral and longitudinal sinuses are invaded. Many cases of lateral sinus thromboses have probably been saved by prompt and radical interference. The sinus has been opened, the infective thrombus removed, and abscesses in the temporal lobe and in the cerebellum drained. Often as a preliminary measure the mastoid antrum and the middle ear have been surgically dealt with, but when there is evidence of sinus thrombosis it is a waste of time and opportunity to stop at this step.

Local applications of heat and cold to the head may be used, and serve sometimes to relieve the headache and modify the delirium. In view of the septic nature of the disease, supportive measures are emphatically indicated from the first. Against the septicæmia we may bring measures to bear that favor elimination by the skin, bowels, and kidneys. The administration of antiseptics, such as the salicyl preparations and the mercurials, are of doubtful value, but are strongly advised by many and should not be omitted. In protracted cases autogenous vaccines may be of service.

## CHAPTER IX.

### CEREBRITIS, ENCEPHALITIS, AND ABSCESS OF THE BRAIN.

INFLAMMATION of the brain proper may be *local or generalised, acute or chronic*.

**Acute Localized Cerebritis.**—**Etiology.**—The brain is subject to inflammatory processes the same as any other parenchymatous organ, yet *generalized cerebritis* is rare, and only recently have we learned to recognize it. *Localized acute cerebritis*, on the other hand, occurs with frequency, but is practically, if not invariably, a secondary condition. *Meningitis* always entails some underlying cerebritis. A circumscribed cerebritis occurs about a *haemorrhagic focus* or spot of thrombotic softening if infection is added. Without the additional infection such as is furnished by septic endocarditis or other infective focus the zone surrounding a cerebral tumor, hemorrhage, or area of softening is one of congestion, edema, and pressure necrosis. *Blows on the head* may cause localized meningitis and cerebritis at the point of violence, or on the opposite side of the head by the action of *contre coup*. In some cases the local cerebritis alone follows, and this may only affect the white matter below the cortex. Apparently the transition favors the local action of bacteria by reducing the resistance of the tissues, as proved by Elsmoother in an interesting series of experiments.<sup>2</sup> Cranial fractures, punctured wounds, and perhaps severe concussion may cause it. Bone disease, septic processes in the diploë, and in the cranial body and

<sup>1</sup>—Boston Med. and Surg. Jour., 1902.

<sup>2</sup>—Rev. Neurolog., Aug. 1906.



venous sinuses, may lead to cerebral inflammation, and be followed by abscess. Infection of the brain after *specifics* sometimes causes a diffuse cerebritis that may involve an entire hemisphere. *Hæmii cerebri* is generally a manifestation of a septic inflammatory process. *Stroking*, *head-stroke*, and alcoholism seem capable of inducing cerebritis, or at least of so modifying the cerebral nutrition that demyelitis may follow and sclerotic brain-tissue be found postmortem. To Van Gieson we owe a knowledge of the cellular changes following involution and their probable relation to an acute auto-intoxication.

In certain conditions the inflammation falls on definite cerebral structures. In this way arise the clinical symptom groups described under the terms *acute poli-encephalitis superior* and *acute poli-encephalitis inferior*, when the cranial nuclei are invaded in the upper or lower groups, furnishing acute nuclear ophthalmoplegia in one instance and acute bulbar palsy in the other. These have already been described with the diseases of the cranial nerves. (See page 155.) Strümpell alleged an acute inflammatory process in the cellular cortex, especially of the motor regions, analogous to that of poliomyelitis as the explanation of many cases of cerebral palsy in children. To this he applied the term *polio-encephalitis*, a name given also to the inflammations of cranial-nerve nuclei, making an additional adjective necessary. It is, therefore, sometimes called *polio-encephalitis anterior* or *corticalis*. All of these *polio-encephalitides* are analogous to the spinal form or identical with it and often clinically associated.

The pathological anatomy of the various conditions which have been assembled under the head of acute localized cerebritis shows at first a high degree of vascular engorgement, edema, and punctate hemorrhage. The inflamed tissue presents above the level of the surrounding part, and is often slightly softened. Its reddish color and yielding consistency have led to its description as a red softening. It has much the macroscopical appearance of embolic red softening, with which it was long confused. Leukocytic infiltration of the brain and the disintegration of neuroglia, myelin, and nerve-cells may induce a pulsatious and even creamy consistency. A nidus ripe for infection is formed, and when this occurs abscess formation is prompt. Later on, if not infected, there is usually more or less shrinking of the inflamed mass, absorption of the necrosed elements, proliferation of the connective tissue, and sclerotic thickening which may reach a leathery toughness. Small foci may in this way become cicatrized. Finally, after years, it may be impossible to say whether inflammation, hemorrhage, or thrombosis was the initial feature of the cerebral defect.

**Symptoms.**—The indications of acute localized cerebritis are obscure. Often associated conditions, such as meningitis, overshadow it in the clinic. Whatever be the seat of the cerebral inflammation, there is usually headache, sometimes vomiting, and rarely optic neuritis. Delirium or somnolence may be present or may alternate. Involvement of special sensory or motor convolutions or pathways gives rise to localizing features marked by defect or disturbance of function. The clinical picture, therefore, always strongly suggests meningitis.

**Acute Hemorrhagic Encephalitis.**—Strumpell, in 1883, and shortly afterward Leichenstein, in 1890, called attention to cases of diffuse hemorrhagic encephalitis. In 1895 Oppenheimer<sup>1</sup> reviewed the literature of the subject, pointed out the intimate relation of the disease to Wernicke's *polio-encephalitis superior* and to Strumpell's *polio-encephalitis anterior*, and added six cases, several of which recovered. Other cases have been reported by Fürstinger, Putnam, Freyhan, Newerk, and Brice, all of which are quoted by Putnam.<sup>2</sup> Brice<sup>3</sup> has since reported a second instance, with full autopsy and bacteriological investigation. There can be little doubt that numerous cases have been mistaken for meningitis. Anatomically the disease is marked by multiple, non-suppurative, inflammatory foci showing congestion and punctate or massive hemorrhages, leukocytic infiltration, and localized destruction of brain-tissue.

**Etiology.**—Regarding the causation of acute hemorrhagic encephalitis there is much to be discovered. Most of the reported cases followed influenza. Putnam's last case followed the mumps, and erysipelas, diphtheria, typhoid, typhus, and malaria<sup>4</sup> have been followed by localized hemorrhagic encephalitis. The writer has seen two cases after influenza, one after pneumonia, and one with some unknown infection associated with acute nephritis. It is a matter of speculation whether various infections act locally or by their elaborated toxins. Southam and Keen<sup>5</sup> found the *staphylococcus aureus* in five of six fatal cases in man, and experimenting on guinea-pigs, produced similar brain lesions by inoculations with pure cultures of this bacterium. An interval, a sort of incubation period, is often noted between the infectious disease and the manifestation of the cerebral symptoms. Children and young adults furnish most cases.

**Morbid Anatomy.**—The meninges commonly are normal in appearance. There is usually some increase of cerebral fluid, and the ventricles may be largely distended by blood-tinged contents. The choroid plexuses and the vessels generally are engorged. Scattered throughout the brain, but principally in the white matter of the hemispheres and in or about the basal ganglia, are foci of hemorrhagic, softened, infiltrated brain-tissue. In some instances the hemorrhage is massive, in others there is but a slight hemic coloring. Microscopically the blood-vessels are found distended, the lymphatics filled with blood-cells and leukocytic elements, and at various points they are ruptured into the surrounding brain-substance.

The influenza bacillus has sometimes been found in these foci,<sup>6</sup> and there are some who think the process may originate in bacterial embolism.

**Symptoms.**—The symptoms of acute primary hemorrhagic encephalitis are not definite, and commonly suggest meningitis. A preceding acute infection may be followed immediately or after several weeks by

<sup>1</sup> *Deut. Zent. f. Nervenhellk.*,<sup>2</sup> 31, vi.

<sup>2</sup> *Jour. Nerv. and Ment. Dis.*,<sup>3</sup> Jan., 1897.

<sup>4</sup> Dana, *Medical Record*,<sup>5</sup> July 7, 1900.

<sup>5</sup> *Amer. Jour. Med. Sci.*,<sup>6</sup> March, 1905.

<sup>1</sup> *Neural Congress*,<sup>2</sup> No. 1, 1897.

<sup>3</sup> Putnam, *loc. cit.*



headache, headache, vomiting, convulsions, or localized palsies. Sluggish pupils and opuntis are rather common. Sleepiness that tends to coma is usual, and rigidity of the neck has been repeatedly observed. The pulse may be slow, the temperature elevated, normal, or subnormal. The palsies, which may be multiple, declare the localized processes and have a tendency to vary in intensity from day to day. Pulse and respiration become shallow and frequent, the stupor deepens, before may form, and death terminates the case in a few days or in several weeks. A fair proportion of cases, as insisted upon by Oppenheim and others, recover, almost, if not completely, after a tardy and anxious convalescence.

Following the pandemic of influenza of 1916-1919 a sprinkling of these cases was observed over a world-wide area. The minority of them were preceded a few days or weeks by definite attacks of influenza, but most of them developed without such precursor. They were symptomatized particularly by oculomotor pareses, and less frequently by other cranial nerve disturbance, by apathy, and somnolence frequently reaching a comatose depth, sometimes by stiffness of the neck, the Kernig and toe signs, by fluctuant temperature, and rarely by a pleocytosis of the spinal fluid. The mortality was high, perhaps one-half terminating in a slow recovery, not infrequently marked by persistent motor and mental defects.

They were variously denominated *lethargic encephalitis*,<sup>1</sup> *sleeping sickness*, *mona*, a term revived from the epidemic of 1890, and, perhaps best of all, as *epidemic encephalitis*.<sup>2</sup>

Edema congestion, and minute hemorrhages mainly affecting the brain stem, basal ganglia, and centrum ovale, were the main findings. Cultural examinations and tissue searchings were practically negative.

It will be seen that the disease is clinically obscure and the diagnosis, except in the epidemic cases, has usually been made post mortem. During life it is usually confounded with meningitis. This is not of great practical moment, as the treatment is the same. The prognosis is grave, but rather more favorable than the early descriptions indicated. Gradual onset, comparative mildness of symptoms, and long duration of disease have a favorable import.

**Chronic cerebritis, and atrophic encephalitis** are terms loosely applied to late and usually secondary conditions that are mainly sclerotic and degenerative in nature. This sclerosis may exist in disseminated patches or in large, circumscribed areas; it may involve an entire hemisphere or be largely confined to the gray matter of both half-brains. The portion of the encephalon thus affected is disturbed in its function, which is usually greatly impaired or entirely abolished.

**Abscess of the Brain.**—Brain-abscess is always secondary, and is due to the invasion of pyogenic bacteria.

**Etiology.**—In a rough way we may say that all the causes of infective sinus thrombosis and acute localized cerebritis are competent to produce brain-abscess, as the first step in its development is inflammation. *Suppurative otitis-media* furnishes almost two-fifths of the cases,

<sup>1</sup> Kossovsky, "Wien. klin. Wochenschr.," July 26, 1917.

<sup>2</sup> Bannet, P., "Ann. Anat. Med. Assoc.," April 3, 1919.

and head injury about one-fourth. About one-sixth of the cases are due to *general pyæmic states*. Of this group purulent pulmonary diseases furnish the great majority. Septic inflammation in the nasal and pharyngeal spaces, brain-tumors, infected cerebral hemorrhages, and infarcts furnish a small contingent. In rare cases the *ædium alticans* has been found in the abscess and traced through the blood-vessel into the nose. In others the only pathogenic bacterium present is the *tubercle bacillus*. *Actinomyces* have also been encountered. Males are about three times as subject to abscess of the brain as females, and five times as frequently affected with the traumatic variety. More than half of the cases occur between ten and thirty years of age. Koenig states that in Prussia about five per cent. of all deaths between the ages of ten and twenty are due to cerebral complications of otitis, mainly abscesses.

**Pathological Anatomy.**—The most common seat of brain-abscess is in the temporosphenoidal lobe, due, doubtless, to the relation of the venous circulation of this part of the brain and of the middle ear with the petrosal sinuses. In decreasing frequency follow the cerebellum, the centrum ovale, the pons, the occipital lobes, the parietal lobes, and the frontal lobes. The *carotid pathway* is often obscure, but the arterial route has been demonstrated in some instances, the perivascular spaces in others. The common sequential relation of middle-ear disease, sinus thrombosis, and brain-abscess calls attention at once to the venous channels as entry-way for the pyogenic bacteria. The extension of cerebral phlebitis from a sinus thrombosis was repeatedly mentioned in the preceding chapter as inducing cerebral softening and abscess formation. Frequently the abscess is *single* and of a size varying from a pea to a hen's egg or even to a larger size. In the regions of latent lesions, especially in the frontal and occipital lobes, an abscess may attain very large dimensions and contain many ounces of pus. Very commonly *multiple* brain-abscesses are encountered. In ear disease, abscesses, both above and below the cerebellar tentorium, are generally encountered, and failure to explore the cerebellum after the examination of an abscess in the temporal lobe has lost lives. When the infection arises from purulent lung disease or septic endocarditis, the left side of the brain is more affected for the same reason that embolism favors the left hemisphere. Under these circumstances, and in the case of infection from typhus, enteric fever, and other general pyæmic states, numerous small abscesses may be found. In this way the brain may be fairly riddled with multiple abscesses. Relatively the grey matter of the brain is less liable to abscess formation than the white substance, and the cortex is often preserved over an extensive underlying abscess. Rarely a fibrous tract communicates with diseased bone, or reaches the outer surface of the cranium, or discharges into the nasal fossæ. On the other hand, no connection may be discernible between the infection and the abscess. The *pus* varies with the nature of the infection and the age and character of the abscess. In some recent cases it merely saturates the softened tissues; in others it is encysted; in a third variety an encysted old abscess is found floating in a secondary, surrounding, more recent



abscess, the walls of which are made up of infiltrated, purulent, softened brain-tissue. In the old cases the pus is thick, yellowish or greenish, and in about a third of them extremely fetid and offensive. Sometimes it is reddish from the admixture of blood. The pus-corpuscles and leukocytic elements vary according to the age of the lesion. The streptococcus is usually found, but pneumococcus, staphylococcus, bacillus pyocyaneus, bacillus tuberculis, and the bacteria of various mixed infections have been noted.

The majority of abscesses are *cystoid*. When located near the surface, the meninges may form part of the containing wall. A beginning cyst-wall has been noted as early as the thirteenth day. Its thickness and consistency increase with age. It is made up by a proliferation of the neuroglial tissue and the fibrous structures of the brain. Usually delicate, in some instances it forms a glistening, resisting membrane of almost a horny consistency, and it may even calcify. The brain-tissue surrounding an abscess is usually more or less softened, and if infected rapidly breaks down, leaving the old abscess-cyst alone. Rupture of an abscess by its steadily increasing contents, which is the usual ultimate accident, inundates the brain-tissue or breaks into the meninges, setting up a purulent process, or floods the ventricles and promptly terminates life.

**Symptoms.**—The symptoms of brain-abscess are usually indefinite, often very obscure, and sometimes entirely unnoticed by patient and physician. Different cases present the widest variety of clinical phases, depending upon the virulence of the infection, the rapidity of abscess growth, the location of the process, and the complications. Some cases run a rapid course to a fatal termination in a few days, and others present a latent period that may last months and even years, to end with rupture and death. We may, in some cases, make out three stages,—one of invasion, one of remission, and a terminal one of paralytic features. In other words, one of cerebritis, one of encapsulation and latency, and one of rupture, infection, meningitis, or ventricular inundation, and death.

The *invasion stage* presents the obscure picture of encephalitis. There is a low febrile movement marked by a vacillating temperature, which sometimes has a distinctly subnormal tendency, with a slow pulse. An intense and persistent headache lasting days, taken with the temperature, suggests meningitis. The headache is often circumscribed and may correspond to the location of the abscess, but as frequently is felt at a distant point. Rigors and profuse sweats indicate the septic character of the disease. A varying degree of leukocytosis is frequently found. Vomiting and constipation or diarrhea are often present. Papillitis or optic neuritis only rarely occurs. The mental state is one of torpor and indifference, or delirium may be present and the sharp "hydrocephalic" cry may be uttered. This stage lasts from two to ten days and may pass at once into the third stage or be followed by a remission.

The *remission stage* is gradually established by the subsidence of the disturbances that appear in the period of invasion. The headache, fever, vomiting, and mental irritation almost disappear or entirely cease. Occasionally the improvement is very prompt and complete. A *latent*

period is thus produced that may extend almost indefinitely. The gradual increase in the size of the abscess and its encapsulation gives rise to very little disturbance. Its effect is that of a foreign body, and its presence is marked by much less disturbance than attends the growth of a solid tumor. At times there may be intense headache, vomiting, and even convulsions. An occasional temperature, or a tendency to a remitting temperature, *erectus*, and emaciation may show the hectic state and alone indicate the ever-present danger.

The *pyrexial stage* is the usual termination of brain-abscesses that have presented a period of latency or remission. It is of sudden onset, and rapidly runs its course within a few days. It not infrequently promptly follows the invasion period, and the fatal mechanism is usually the same in both. In some cases this consists of a rupture of the abscess, causing a rapid infiltration of the cerebral capsule; an invasion of the medulla; a tearing through the cortex, setting up a purulent meningitis; or the inundation of the ventricles. There remain, however, a large number of cases in which these anatomical conditions are wanting to explain the sudden onset of this stage. Ordinarily an apoplectic stroke, with or without convulsions, takes place. Usually there are no premonitions or only the vague indications of cerebral mischief which have presented at intervals during the latent period. The apoplectic coma may be punctuated by Jacksonian fits, marked by conjugate deviation of head and eyes and some lateral weakness, and the patient may succumb in a few hours without regaining consciousness. Death, indeed, may be sudden. If the immediate effects of the stroke subside, a hemiplegia with marked rigidity, and often with spasmodic features, is progressively developed. The headache, the fever, and the symptoms of the early stage reappear or become intensified. Nystagmus, pupillary inequalities, and involvement of the ocular muscles appear and death is likely to follow, preceded by delirium and coma.

The *healing symptoms* of brain-abscess are rarely prominent. Usually they are quite indefinite or entirely lacking. The location is often in the frontal, temporal, and occipital lobes, or in a cerebellar hemisphere, whence focal symptoms do not arise. As a rule, an abscess of the brain, due to disease of the cranial bones, lies subjacent to the original lesion. In the same way ear disease gives rise to abscess near the petrous, but in many instances the purulent collection has been found at a considerable distance, and even in the opposite half of the brain. Abscess due to embolic process from the heart or lungs is likely to be located in the capsular or Sylvian arteries, and gives rise to early hemiplegic features or disturbances of the motor cortex. The circumscribed headache sometimes furnishes a localizing feature, but it can not be relied upon. MacEwen has noted a higher percussion note over the abscess than was yielded by the rest of the head, and Dana has confirmed this symptom in one case. Local tenderness and increased temperature may occasionally be made out. When the abscess is associated with septic sinus thrombosis, we have, in addition, the local superficial evidence of that disease. Even when the sensorimotor zone is



involved, the symptoms may be very slight. Abscesses may, however, yield as definite localizing features as any other encephalic lesion, and these then have the significance and value discussed under the head of Cerebral Localization. Involvement of cranial nerve-trunks, cerebellar symptoms, hemianopsia, Jacksonian fits, aphasia, and other localizing indications should always be carefully sought. Multiple abscesses also lead to symptoms of corresponding diversity.

**Diagnosis.**—The diagnosis of cerebral abscess depends very largely upon the history or presence of trauma or of an infective condition about the head and face or in the thorax. A chronic otitis, maxillary abscess, otitis, sinus suppuration, or purulent pulmonary disease, followed by headache, vomiting, delirium, stupor, slow pulse, vacillating temperature, and rigors, means encephalic invasion. These symptoms are common to meningitis and abscess, and the differential diagnosis can not always be made. Any evidence of a circumscribed process, however, favors the idea of abscess, and hence localizing symptoms become very important. The two conditions are often associated and a terminal suppurative meningitis is common in abscess. In later periods the differentiation of cerebral abscess from a growth may be impossible; nor is it essential. The terminal stage, with its rapid onset and paralytic features, may readily be confounded with cerebral hemorrhage or softening if it occurs in middle life and is preceded by apparent health. When systemic sepsis is manifested, the purulent character of the encephalic process is less doubtful. A marked leukocytosis is of important diagnostic and prognostic significance.

Abscesses resulting from ear disease, bone disease, and disease about and in the facial cavities—in other words, abscesses due to direct invasion—are ordinarily single. Abscesses resulting from pyemic states and from purulent thoracic conditions are usually multiple. Cerebellar abscesses are also usually multiple. The localizing diagnosis is made on the lines already laid down in Chapters III and IV of this part.

**Prognosis and Treatment.**—Suppurative disease within the cranium is always grave. Although a cerebral abscess may encapsulate and lie dormant for years, this can not reasonably be expected to take place, and even when it does, it constitutes a constant menace to life. Encapsulation does not necessarily check suppuration, and eventual rupture or secondary infection is the legitimate sequel. The late results are ordinarily fatal, and latent cerebral abscesses account for a certain proportion of sudden deaths.

The treatment of cerebral abscess should be prophylactic. Chronic suppurations in ear and nose should never be neglected and the utmost precautions should be taken, in the management of all wounds about the head and face, to prevent infection and sepsis. From these largely controllable sources the great majority of brain-abscesses and other endocranial suppurations arise. When the diagnosis is established, there should be immediate recourse to surgery. Of all encephalic diseases abscess promises the best results to surgical measures. Adequate drainage after opening the skull has been followed by brilliant success in numerous cases and in the hands of many operators. All other measures are inadequate.

## CHAPTER X.

## THE CEREBRAL PALSIES OF CHILDREN.

A SUMMUM of varied developmental and acquired cerebral defects result in paralysis in childhood. They involve or destroy within the cranium some portion of the upper motor neuron, and give rise to clinical types that justify a separate description. Etiologically, they may be divided into three groups: (1) Those due to prenatal conditions; (2) those following birth accidents; and (3) those dependent upon disease or trauma after birth. Clinically, we also divide them into those showing unilateral defects, the hemiplegic cases, and those presenting bilateral defects, the diplegic cases.

**Etiology.**—One of the most important *perinatal conditions* resulting in infantile cerebral palsy is an actual deficiency of brain elements, a true agenesis. This may be marked by convolutional simplicity and a lessened number of cortical cells and pyramidal fibers. In other cases a part or the whole of a hemisphere, or of both hemispheres, may be lacking. Between these gross morphological defects and a condition in which the neuron units have diminished dynamical qualities or a lessened power of endurance, that in early life leads to their atrophic or sclerotic degeneration there is but a difference of degree. The lack of constitutional endurance, of capacity for growth, and of resistive power underlies many brain and cord diseases that present hereditary and familial characteristics. In other instances it furnishes a lowered resistance to toxic influences. In given cases unfinished children, lacking motor elements in the cortical mantle, and children born prematurely, before the pyramidal apparatus has been well established, present various degrees of motor and cerebral defect. The upper motor neuron is well formed only at the ninth intra-uterine month, and not completely developed until the second or third month after birth. Much defect in the upper neuron is always marked by spasticity and impaired motility in the muscles supplied by the lower or terminal motor neuron.

Rarely direct traumatism has affected the brain of the unborn child which presents hemiplegic contractures at birth. In other cases evidence of hemorrhage and softening has been found, and in many instances the probability of a prenatal meningitis-encephalitis has been upheld by the presence of localized and diffuse sclerotic changes in the brains of newborn children. Inherited syphilis and toxic conditions due to illness of the mother have been accused in some of these cases. Perencephalia is usually due to intra-uterine disease of the cerebral vessels.

The conditions attending birth frequently lead to brain-lesions in the child. The great majority of cerebral birth palsies occur in protracted labors, and consequently in primiparae. A number of



them follow precipitate labor and in both are due to violent compression of the fetal head. Comparatively few can be attributed to the use of forceps, and it is exactly in these cases that the labor is likely to have been tedious. Forceps accidents, however, can not be denied or overlooked, and the misuse of these powerful instruments is fraught with serious results to the skull and brain of the child. The facts, however, favor early skilful instrumental interference as compared with tedious labor.

The frequency of hemorrhage into the cerebral and spinal meninges during birth has been put on a sound foundation by the investigations of Litzmann, McNutt, and Spencer. It is found in the great majority of stillborn children and is the common cause of asphyxia of the newborn. Occasionally this blood comes from a ruptured longitudinal sinus, but most frequently from the pial vessels. Punctate and larger hemorrhages within the substance of the brain and cord are frequent. The results of these birth hemorrhages depend upon the quantity and location of the effused blood. Where death does not follow, all degrees of disability are encountered. Palsies follow the involvement of the motor cortex or its pyramidal tracts, but if the frontal lobes are seriously affected, idiosyncrasy is a consequence and is a frequent accompaniment of motor disturbances. Similar disturbances of the latent lesion territories of the brain may give rise to insignificant or very obscure symptoms. As a rule, the hemorrhage is basilar in location in vertex presentations and vertical in breech cases. The absorption and organization of these hemorrhages lead to a more or less diffuse sclerosis of the brain-substance that inhibits its growth and diminishes its functional capacity. In other cases in terminal stages large areas of softening and cyst-like formations are found that are indistinguishable from porencephalia. In about one-fourth of these cerebral birth hemorrhages the spinal cord is similarly affected. Some cases of syringomyelia may have this origin.

The *postnatal* causes of cerebral palsies in children, according to Osler's list, are hemorrhages, embolism, endo- and peri-arterial changes, encephalitis, and cerebral venous thrombosis. In other words, they are the same as in adults, but hemorrhage is more frequent than thrombosis. The relation of acute infectious diseases to embolism and arteritis only needs to be recalled. Traumatism and tumor formation cause some cases. Of encephalitis much has been said, and Strumpell claimed cortical polio-encephalitis to be the lesion in many of these cases, but the exact pathological process still escapes us owing to the paucity of early post-mortem examinations. Cerebritis in infancy and childhood is a more frequent occurrence than formerly supposed. The lesion is often primarily vascular. Gowers was the first to insist on the part played by venous thrombosis, and others, among them Osler, have added weight to the contention. This cerebral venous thrombosis is often a part of sinus thrombosis, to which it may hold a primary or secondary relation. It serves to produce softening, and sclerotic changes in the cortex and palsies follow. As many of these palsies start with convulsions, the question arises whether the convulsion is the cause or effect of the lesion. It may be either. There can be no doubt that the

increased arterial tension of the convulsed state may cause rupture of cerebral vessels, but, as a rule, the convulsions are due to the irritant effect of the lesion. Later on epileptic or epileptoid convulsions are very commonly encountered. Sex plays no etiological part. Males and females are about equally affected. One-half the postnatal cases in children occur within the first three years of life.

**Merbid Anatomy.**—The post-mortem findings in these cases furnish various lesions. Sachs gives the following table:

CLASSIFICATION OF INFANTILE CEREBRAL PALSIES.

TUMORS.	Meningeal Lesions.
I. Prolapsus of intra-arterial coarct.	Large cerebral defects, porencephalia. Defective development of pyramidal tracts. Agensis cerebelli, highest nerve-centers involved.
II. Birth palsies.	Meningeal hemorrhage, rarely intracerebral hemorrhage. Later conditions: Meningo-encephalitis chronica, sclerosis and cysts, partial atrophies.
III. Acquired palsies.	Hemorrhage, meningeal and rarely cerebral. Thrombosis, from endarteritis, and in traumatic conditions. Embolism. Later conditions: Atrophy, cysts, and diffuse and lobar sclerosis. Meningitis chronica. Hydrocephalus addens the sole case. Primary encephalitis and the poly-encephalitic verticillate areas of Striepenell.

The difficulty of deciding the origin of the terminal conditions is very great and it is often impossible. Porencephalia, for instance, may be due to defective development, to embolism, to thrombosis, or to hemorrhage. The initial process of a diffuse sclerosis may be hemorrhage or inflammatory.

**Symptoms.**—The clinical history of cerebral palsies in children varies in the three sets of cases. In the prenatal cases the condition is congenital, but may not be noticed for some time after birth. In birth palsies the condition is usually noticed shortly after birth or develops within a few weeks. In the later-acquired cases the patients may present an ordinary record up to the onset of the paralytic features. In their final development all present very common attributes and are usually indistinguishable by external examination alone. The great majority of natal and prenatal cases have bilateral palsies. After birth the tendency is to one-sided paralysis and after the age of three it is almost the invariable form. A pure cerebral monoplegia is so extremely uncommon as to be almost unknown.

**Hemiplegic Cases.**—The hemiplegic cases are the best type for study, as the unaffected side furnishes an opportunity for comparison. Ordinarily the paralytic features develop after an acute febrile attack, after or during an acute infectious fever, or in traumatic states. The



child is seized with convulsions, which are more severe on or entirely confined to one side of the body and to the side that is afterward paralytic. The convulsive attack is usually prolonged, lasting for several hours or even for a day or two, and perhaps returns several times within a few days. During the convulsive seizures the temperature may be markedly elevated and more or less unconsciousness is commonly present. The child is left weak and exhausted, often with continued feverishness, and the loss of power on one side is frequently only incidentally noticed. If the child had formerly spoken, speech, as a rule, is temporarily lost, whether the paralysis be left- or right-sided, but after the age of about six years aphasia is well marked only in lesions of the left side in right-handed children. If their intellectual faculties are not destroyed, speech even then is regained with surprising rapidity.

The paralyzed limbs soon develop *marked spasticity* with *exaggerated reflexes*, which the spasticity may conceal unless the examiner be attentive to the play of tendons when the tests are made. The spastic condition is also followed by *contractures* which place the limbs in the positions so characteristic of hemiplegia in adults. Here also are similar *attitudes and gait*.

*Sensory disturbance* is apparently absent even in the recent cases, and *electrical reaction modifications* are never present. The *trophic condition* of the paralytic limbs, however, is reduced, as is shown in the unequal growth on the two sides as time goes on. This results in *some deformity*. The shoulder girdle is smaller on the affected side, the chest and arm less in size, and the pelvis and lower extremity unequal to corresponding parts of the sound side. The half of the head and face on the paralytic side may be of inferior dimensions. The limping gait of hemiplegia is increased by the shortened limb, the pelvis tilts, the spine becomes *scoliotic*, and the retarded, paralytic, contracted upper member is held to the side of the body and usually fixed in all its joints.

From nine months to two years after the paralytic onset the paralyzed side, especially the hand, in over one-half of the cases, is animated by automatic and involuntary *choreoid or athetoid movements*, which often attain a wonderful complexity and range. They may also involve the face, but seldom to the degree that is observed in diplegic cases. These athetoid movements are usually intensified upon voluntary effort to use the limb. Attempts to grasp an object will often cause the fingers to move widely apart in extreme extension, and after clumsy, slow movements the object is awkwardly and insecurely held or the attempt fails. In some cases the extremities, particularly the upper one, are writhed about in the most vigorous, serpentine, and purposeless way, striking the patient's face or getting into awkward positions behind the neck or back.

The athetosis in rare cases is persistent day and night, in others it subsides during sleep, and in still others, and perhaps the majority of cases, it only appears when provoked by voluntary effort or emotional disturbance. In the cases that are marked by excessive athetoid mobility the muscles, from constant though involuntary use, are frequently firm and well nourished. They may even be over-developed,

Clark<sup>1</sup> has proven hypertrophy in such cases and actual enlargement of bones has been shown by the x-ray. On the other hand, well-marked atrophy may be encountered. The joints frequently present a



Fig. 30.—Attitude and deformity in removed right hemiplegia in a boy of seven.



Fig. 31.—Right hemiplegia—stiff arm both with extension in right side of face and stiffness of right leg.

marked increase in their range of motion so that the digits, for instance, can easily be laid on the dorsal aspect of the hand. This condition is particularly marked in the severe cases, beginning very early in life. The unclenched and tentacle-like movement of the fingers and toes in athetosis, once recognized, can hardly be mistaken for anything else. Associated movements by which the paralytic member apes the position and motions of the sound fellow reach their highest development in these cases in children.

In the hemiplegic cases the arm usually recovers less than the leg or face and the athetoid condition is commonly confined to it. Contractures predominate in the foot and leg, producing an equinovarus, rarely a valgus, deformity, which is also figured by the shortness of the limb, and there is some tendency to contracted flexion at the knee and hip. The toes are frequently cramped and distorted by the contractures and hammer-toe is a usual deformity, but when athetosis is present an abnormal range of passive and active motion is found. In the face the tendency to contracture is frequently only shown in an exaggeration of the facial muscles during emotional expressions as shown in figure 91, the boy being pleased at having his picture taken. Any degree of

<sup>1</sup> *Jour. Nerv. and Ment. Dis.*, Nov., 1902.



mental enfeeblement may be present in hemiplegic cases, but usually it is much less marked than in the bilateral form and may be practically



Fig. 96.—Infants—standing from birth. Right arm and right leg stiff and extended.



Fig. 97.—Infants—standing from birth. Right arm and right leg stiff and extended.

absent. The tendency to *epileptic attacks* and mental deterioration is pronounced in the hemiplegic form, and will be mentioned again.

**Diplegic Cases.**—In the bilateral form the unilateral conditions of the hemiplegic cases are present on both sides, but the lower limbs are, as a rule, more strongly affected than the upper and the face may show little or no motor impairment. In rare cases the trouble seems to be limited to the lower limbs, and these instances are sometimes described as *paraplegic*. Almost invariably, however, the presence of speech difficulties, of mental defect, and of clumsiness in the use of the hands will betray the diplegia. The frequency of spinal hemorrhages in stillborn children, as determined by Spencer, for instance, gives ground to suppose that in very exceptional instances the conclusion may alone occur and a true paraplegia result if the child survives. These bilateral cases are mainly of prenatal and birth origin, only a small number arising after the first few months of infancy and practically none after the third year of life. The more extensive injury to the brain is attended almost of necessity by greater mental impairment and blinder marks many cases. Microcephalia sometimes results or the skull may have so defective a shape that *potercephalia* and *anencephalia* may be suspected. Such children are either noticed to be rigid and insensitive at birth, or after birth-injuries and convulsive manifestation develop rigidity and contractures during the early months of infancy. Frequently it is only when the child is found incapable of learning to sit up, or to use its legs in

efforts at walking that the paralytic state is recognized. Mental backwardness, slowness in the development of speech, and other indications of injury to the highest brain functions are often neglected until the third or fourth year in the misguided hopefulness that the child will "outgrow it." The rigidity and spastic state is frequently so great that the legs and arms present a "lead pipe" resistance to passive move-



FIG. 24.—SPASTIC DIPLEGIA.

ments. Voluntary efforts are hindered or defeated, and some of these children never get the hands to the face, much less their toes to their mouths. They are difficult to handle and to dress. Their arms and legs are as unmanageable as stiff-jointed manikins. They are "all thumbs" and awkwardness. The spasticity in the lower extremities, which tends to flex hips and knees and especially to adduct the thighs, holds the knees closely together, makes it difficult to dress and bathes them as infants and defeats locomotion later on. Placed on a chair, the lower limbs have a tendency to maintain a rigid horizontal position. If they are placed on their feet the legs cross, the heels can not be brought to the floor, and if steps are taken it is only by advancing the foot that is in front and then bringing up the one in the rear. As they grow older a tendency to equinovarus or varus and genu valgum is induced, but by keeping the feet widely separated they are able to dodge one knee around the other. They thus advance in a shuffling, knee-rubbing, toe-scraping, laborious manner, to which the increased reflexes and ankle-clonus give a jerky, tremulous, spastic character.

In some cases more or less athetosis is present in all four members, and occasionally it invades the face and even the tongue, pharynx, and larynx. When the athetosis is general, and particularly when it involves the feet, the patients are rendered almost completely helpless physically.

**Little's Disease.**—English, German, and American writers are prone to use the term Little's disease in a generic sense to embrace all cerebral palsies in childhood. French writers, of the Salpêtrière school especially, insist upon a clinical form of spastic diplegia to which they give the distinctive name of Little's disease, after the London surgeon who among the first called specific attention to this class of disorders.



As these cases referred to present certain definite characters and a more hopeful future than the others, they merit separate mention.

The initial condition is a premature birth or the birth of a markedly undeveloped child. This means a defective pyramidal tract in the spinal cord and brain. Such children usually weigh less than four pounds. Spasticity results in the territory of the lower neuron from the lack of control due to the undeveloped condition of the upper neuron. These children are rigid from birth, but the mental qualities are not necessarily diminished, and if they survive, the tendency is to continuous improvement as the pyramidal tracts develop. This development is never complete, but improvement continues up to the age of full growth. The motor condition is one of pure spasticity and there is no tendency to athetoid movements or epileptic attacks. The gait remains more or less spastic through life, which may be a long one marked by ordinary or even brilliant mental activity.

**Amaurotic Family Idiocy.**—Sachs<sup>1</sup> propose this clinical designation for a group of cases of which he collected nineteen occurring in ten families, with three autopsies. Numerous other observations have since appeared in current literature, notably those reported by Collier.<sup>2</sup> These children are born at term and present healthy physical and mental development up to the age of four to ten months, when they become weak, lethargic, and stupid. Ocular symptoms soon appear and blindness due to optic atrophy develops. Frey<sup>3</sup> does not agree with Sachs that the condition is merely genetic, but considers the changes found to be postpartum, presenting for childhood the state recognized as amyotrophic lateral sclerosis in adults. The ophthalmoscopic picture is said to be very characteristic. Bead<sup>4</sup> states that the appearance is not that of pronounced atrophy. The disc is not markedly blanched, but the fovea centralis presents a clear-cut liver-colored plaque surrounded by a halo of grayish-white which does not obscure the retinal vessels. Nystagmus, ocular deviations, and pupillary anomalies are frequently encountered. Autopsical findings have shown convolutional simplicity and cortical cellular degeneration. Sachs<sup>5</sup> reports degenerative changes in the gray matter of the entire cerebrospinal axis and even in the root ganglia. These changes have been attributed to toxic processes, but none of the alleged toxic factors can be accepted as competent in all instances. As many as four cases have been observed in a single family, and the inherent anatomical lack in the brain is not explained by inherited epilepsy, which may, however, produce a very similar clinical picture.



Fig. 35.—Case of Little's disease. Rigidity and trembling from spasticity in a child weighing three pounds at birth.

<sup>1</sup> *New York Medical Journal*, May 20, 1896.

<sup>2</sup> *Brain*, 1899.

<sup>3</sup> *Brit. Arch. Clin. Med.*, 18, 112.

<sup>4</sup> *Jour. Nerv. and Ment. Dis.*, May, 1900.

<sup>5</sup> *Jour. Nerv. and Ment. Dis.*, Jan., 1902.

**Epileptic Attacks.**—One of the most serious features of the cerebral palsy of children is the marked tendency to the development of *epileptic attacks*. These appear in fully one-half of all cases. They are due to the brain-lesion, and usually are most marked in the paralytic limbs, but with a tendency to spread and become generalized. True Jacksonian fits are but rarely encountered. In some cases the initial epileptic attack is at once followed at regular intervals of weeks or months by these seizures. In others they do not appear for a period of years and may be preceded by petit mal attacks. They are always to be expected, and when established constitute a serious menace to the health and mental state of the patient. The ordinary tendency of epilepsy to induce dementia is exaggerated in these already defective brains.

**Diagnosis.**—The diagnosis of cerebral palsy when the condition is well marked should present no difficulty. In the lighter grades the epileptic attacks or the choreal movements are frequently mistaken and the organic basis overlooked. The combination of local symptoms and mental enfeeblement with the motor difficulties in diplegic cases should be sufficiently striking. The hemiplegic distribution of paralysis can tell but one story. In cases presenting paretic features it may require a careful investigation to locate the trouble in the brain. The history here, as elsewhere, is a valuable guide. In addition the preserved electrical reactions, the comparatively normal trophic conditions, and the exaggerated reflexes should readily exclude a cerebral lesion. The teratological defect which furnishes the cases of amniotic family idiosy and the defective development of Little's disease must both be deciphered from the clinical data or the post-mortem findings.

**Prognosis.**—The prospects in these diverse cases vary widely, but it may be positively stated that complete recovery never occurs and that a perfectly normal condition is never attained. In the cases of prenatal origin the prognosis must rest on the observed course of development during the first year of life. Improvement in motor control and the appearance of mental awakening are hopeful indications, but the majority of these cases are doomed to mental retardation or idiosy and more or less physical helplessness. The cases that conform to the type of Little's disease are exceptions. In these the prognosis for mental development is good and progressive motor improvement, up to a certain point, may be confidently expected. Amniotic family idiosy, which is a congenital defect, is, at present, of absolutely fatal prognosis.

In the cases of birth palsy the intensity of the convulsions, their persistence, and the apparent extent of brain injury, in a very rough way, guide the prognosis as to life. If, after a few months, spasticity is still absent and some voluntary motion occurs in the paralyzed limbs, the prospects are much improved. The question of mental development can only be decided by watching the course of events. In the acquired cases the prognosis is much the same as in the apoplexies of adults as far as motility is concerned. The return of motion in the paralytic limb and the absence of contractures permit hope of a fair degree of motor restoration. Speech is usually restored, unless the frontal lobes have been injured and mentality impaired. The loss of sight and more especially of hearing is likely to prevent mental development and to in-



duce idiocy. Whenever epileptoid attacks appear, they constitute a very serious feature and foreshadow dementia. They are somewhat more common in the late than in the congenital cases and in the hemiplegic than in the diplegic palsies.

**Treatment.**—In the birth-palsy cases the nutrition of the child is the first problem, as suckling and swallowing are often impossible. The evidence of meningeal hemorrhage is usually so indefinite that trephining, which has been proposed, can rarely, if ever, be practised. The convulsions must be subdued by cold applications to the neck and head, and by sedatives, such as chloral, bromid, morphin, and by continued chloroformization, if necessary. The bowels should be thoroughly opened, preferably by means of calomel. The same measures are indicated as in older cases of acute hemiplegic convulsions and in convulsions generally.

In the later cases the management of the apoplectic and paralytic states is the same as for adults (see p. 223). From the first, the nutrition of the paralytic muscles and the tendency to contractures must be met by massage, passive movements, and faradism. As soon as contractures appear, the massage and electricity must be used only on the weaker muscles—that is, as a rule, only on the extensors. The use of these measures to increase the strength of the contracting muscles may occasion decided harm. Unfavorable positions of the limbs must be obviated by mechanical measures, if necessary. Such means may do much to retard contracture, to hasten and increase voluntary control, and to favor growth in the paralytic members. Orthopedic appliances and tenotomies in neglected cases sometimes secure much permanent improvement. Transplantation of tendons has lately come into prominence, and in some instances can be resorted to with fair prospects of improvement.

Athetosis may be subdued to a considerable extent by fixation appliances or plaster-of-Paris splints. Hammond reported some cases temporarily much benefited by nerve-stretching, and Horsley has extinguished the athetosis by excising the corresponding brain-centers. In one such case the writer advised the removal of the cortical centers for a very clumsy upper extremity, and the resulting palsy in the member was replaced finally by slight but useful voluntary control. Temporary flaccidity of spastic and athetotic members may be produced by intraneural injections of absolute alcohol. During such periods the child can often gain material control of the muscles otherwise inhibited by the rigidities, and make substantial and lasting improvement, especially in walking. The intradural division of several posterior nerve-roots for the same purpose by Foerster's method has its advocates, and in severe cases is indicated.

When idiocy is present, it is open to educational methods only. The epilepsy that so frequently complicates these cases can be controlled to some extent by bromids or combinations of various sedatives with bromids. When the attacks are of a distinctly Jacksonian character, the question of operation will come forward. Every case must be considered by itself. As in many instances the condition is one of cerebral sclerosis, no operation can do good. If there be a cystic state, exploration and evacuation may be productive of much relief. In some cases

the mere effect of the operation has been temporarily beneficial; is others it has acted by relieving pressure. Removal of cortical centers in Jacksonian fits of this class is very likely to merely displace the initial symptoms and to add to the traumatic conditions already present. Linear craniotomy when the brain is diseased can only be condemned. If it have any place it is in the cases that present closed fontanels at birth with microcephalic heads, the possible result of early synostosis. Sharpe and Farrell<sup>1</sup> report much benefit in a few cases treated surgically by making a large decompression opening in the skull. In any case showing pressure symptoms such an operation or the simpler one of puncture of the corpus callosum by Anton's method<sup>2</sup> may be seriously considered.

## CHAPTER XI. TUMORS OF THE BRAIN.

THE encephalon is frequently invaded by various new growths common to other parts of the body, and by a number of neoplasms that are practically found only within the skull. In addition, new formations arising from the meninges and cranial walls, while not strictly brain-tumors, present symptoms that are identical with lesions of the cortex, and are localized in the same way. The term "brain-tumor" is here taken broadly to cover new formations within the skull.

**Etiology.**—The causation of brain-tumors is an obscure subject. Many of them arise by metastasis from distant morbid foci, as is usually, if not always, the case in tubercular growths, carcinomata, and parasitic cysts and masses. Some arise by intracranial extension from the orbit, pharynx, and cranial bones. *Hereditas*, aside from tubercular and syphilitic cases, plays a very insignificant if not an entirely negative rôle. *Teratogenesis* has probably been too frequently assigned as the inciting cause of brain-tumor, but it undoubtedly is competent in some instances. Thus, continuous symptoms have arisen within a few days of a head injury, and a tumor has subsequently been found at the traumatic site or in the brain beneath. There can be no doubt that even slight head injuries may localize tubercular and syphilitic activity and lead to corresponding tumor growth. Sarcoma and glioma are the neoplasms that seem to be most frequently attributed to trauma, but at the same time they are among the most common of brain-tumors.

No age is exempt from tumor growth within the cranium. The great majority of cases occur in childhood and active adult life. The activity of tuberculosis in childhood, and the prevalence of syphilis in young adults, as well as liability to traumatic influences, may in part account for this. *Males* are somewhat more subject to cerebral tumor than *females*.

**Pathological Anatomy.**—Of all brain-tumors tubercle furnishes the largest proportion. The inflammation of the tubercular process within the brain sometimes gives rise to the formation of large, solitary, isolated, often encysted caseous masses that may be single, but are

<sup>1</sup> *Jour. Amer. Med. Assoc.*, Nov. 29, 1913.

<sup>2</sup> *Anton and Brauer, "Talkersmarkt," Berlin, 1911.*



multiple in about one-half of the cases. Tubercular tumors favor the base and the course of the large cerebral and cerebellar vessels, which recalls the usual propagation of tubercular meningitis. No part of the brain, however, is exempt. They do not destructively invade the brain-tissue, but displace it and act as foreign bodies, causing pressure atrophy. About them may often be found an area of tubercular infiltration and inflammatory activity, especially if they are seated so as to involve the meninges. Three-fourths of the cases occur before the age of twenty. The tumors vary in size from a pea to a hen's egg, or even a larger size, and in number from one to a score.

Sarcomatous growths are next in frequency. Usually they present the distinctive and important character of being more or less encapsulated and sharply separated from the brain-tissue, from which they can, therefore, if accessible, be easily enucleated. As a rule, they are outgrowths of the cranial dura or periosteum, and the majority of them are situated in the basilar region, involving the brain-axis. In comparatively rare instances they infiltrate the brain-tissue, or, arising at the vertex, spread out broadly. They are of rapid growth, and in a given case furnish pronounced, persistent, and uniform symptoms. They present the variations of cellular elements that mark sarcomata elsewhere. As a rule, they are single.

Glioma is peculiar to nervous structures. It arises from the neuroglia and finds its most usual seat in the brain, though it may occur in



Fig. 36.—Sarcoma of right temporo-sphenoidal lobe. A, External surface; B, transverse section. (Rev. N. A. Jones.)

the spinal cord or retina. It presents a redfish, vascular color and a soft consistency quite like that of the brain-substance. Glioma is eminently an infiltrating and a destructive growth. Its outlines are difficult to determine. Owing to its strangulating effect, the center of a glioma is frequently filled with softened debris and fatty-degeneration products, which may become fluid and the growth thereby cystic. Many alleged focal "brain-hypertrophies" have been gliomatous infiltration in fact, and the microscopic examination of "cystomata" has ably discovered

the true nature of the lesion in other instances. The favorite location of glioma is the white substance near the cortex, and it may infiltrate large areas, even the greater part of a hemisphere. Ordinarily it is single.

In consistency they are soft, often highly vascular, and may in some cases be easily mistaken for angioma. Hemorrhages not infrequently occur in them. In other cases a great amount of interstitial fluid gives them a myxoid character.



Fig. 97.—Glioma of cerebellum containing recent and old hemorrhages.



Fig. 98.—Glioma of right hemisphere.

Carcinomata, practically speaking, are found in the brain only as metastatic growths arising from primary cancer in the body organs.



breast, etc., or through direct invasion by extension from orbital or other cranial and facial carcinomatous growths. They are also practically confined to the second half of life. Uncontrolled by the soft tissue of the encephalon, carcinomata rapidly invade the brain, forming ill-defined, nodular, rarely encapsulated, very vascular, and practically inoperable tumors. When arising secondarily from cancer in the body organs, especially those in the thorax, carcinoma of the brain locates by preference in the neighborhood of the great vessels of the base. In other instances it springs from the choroid structures and the epithelial lining of the ventricles, or from the dura and pia mater.

Cysts form tumors in the brain with comparative frequency. An encysted hemorrhage or softened infarct does not ordinarily give rise to progressive or marked irritative symptoms, and need not be here considered. An encysted and latent abscess may, however, produce all the symptoms of a brain-tumor, which in fact it is. Cysts arising from parasites, such as the cysticercus and celinocercus, are rather common in some parts of the world, but are distinctly rare in the United States. Diamond<sup>1</sup> could only find eight reported cases of cerebral cysticercus in American literature. The central breaking down of sarcomata, and especially of gliomata, produces cysts the nature of which may be indeterminate without histological search. Rarer forms are due to ependymal inclusions in embryonic development, producing later in life ventricular cysts or cysts in the posterior portion of the pineal body. Dermoid cysts have also been encountered within and outside the dura, and especially in the cerebellum. Cyst development is usually slow, and the resulting tumor acts as a displacing foreign body. Their diagnosis is important, as they are readily evacuated and the cyst-wall may be removed when situated in a surgically approachable part of the brain.

Syphilitic tumors of a gummatous sort in the brain are more frequent than statistics would indicate. Owing to their partial immunity to treatment and their readily accepted consequences they are seldom reported. As a rule, they spring from the meninges and large vessels, and, when deeply seated, are usually the ingrowth from one of the penetrating folds or vessels. They may be definitely limited or surrounded by softened brain-tissue, and have no tendency to infiltrate. Their usual location is in the hemispheres, especially at the base, or in the pons. They rarely occur in the cerebellum or central ganglia. After treatment their early soft, encephalic, and gelatinous character may be changed to a shrunken, degenerated, and fibrous condition. They are usually multiple, nodular in form, and about the size of a chestnut. They occur as the result of acquired syphilis, and consequently usually in adults at periods varying from a few months to many years after the primary lesion. They are commonly attended by other syphilitic disturbances of the brain, such as endarteritis, meningitis, and cranial-nerve lesions. Of rapid growth, they usually promptly recede to some degree under adequate treatment, but their absolute removal by medication is always questionable.

Many other tumor forms are occasionally found in the brain. *Cerebroma* is an embryological infolding of the gray mantle, which

<sup>1</sup> "Jour. Amer. Med. Assoc.," June, 1899.

becomes occluded, usually in the white substance, and, later, takes on active proliferation, forming a heterotopic tumor. *Fibroma*, *angiosarcoma*, *neurinoma*, *parosteoma*, *papilloma* of the choroid plexus and Pacchionian bodies, *actinomyces*, *lipoma*, *teratoma*, *osteoma*, *cholesteatoma*, etc., merely require mention. The *psithyrus* tissue associated with acromegaly is discussed under that head, but tumors of various sorts, such as adenoma and adenocarcinoma, occur at this point without such association.

**Aneurysms** sometimes give rise to symptoms of intracranial tumor. They arise, especially after middle life, as the result of arterial disease, discussed in chapter v, or they may be due to trauma. Aneurysms of a circoid character and of great extent that produce brain symptoms are occasionally encountered on the meningeal arteries. The cerebral vessels present aneurysms usually of a globular or pyriform shape. Their favorite location is at the base and in the Sylvian fissure,



Fig. 35.—Aneurysm of the basilar artery.

at the bend of the carotids, in the cavernous sinus region, and in the basilar artery, but they occasionally develop on the perforating arteries and may attain considerable size. The formation of *colicky aneurysms* and their relation to cerebral hemorrhage have already been indicated. Occasionally a patient with cerebral aneurysm is conscious of its pulsations and hears the bruit. The bruit may also sometimes be heard by auscultation of the cranium, but vascular murmurs have also been heard in a case of extensive softening and in vascular tumors. The chief symptoms of aneurysm are due to pressure upon the cranial nerves and brain centers. In only about one-third of all cases are clinical symptoms presented that are fairly referable to the aneurysm.<sup>1</sup>

All *brain-tumors* cause more or less circumferential destruction of brain-tissue. This is at a minimum in slow-growing tubercles and reaches its maximum in rapidly growing and infiltrating gliomas. Almost invariably, if the meninges are reached, an inflammatory thickening over the growth is added. In the case of tubercle and

<sup>1</sup> Boodin, "Brain," Oct., 1907.



syphilis a more or less diffuse meningitis is commonly present, sooner or later. The interpolation of a new growth within the resisting walls of the skull means increased intracranial pressure and disturbed vascular conditions. The effects are serious, relative to the intensity of the pressure, the rapidity with which it is established, and the location of the growth. Even a small tumor in the circumscribed space under the tentorium produces pressure rapidly. In the same way, if located near the falx or other resisting structure, its pressure effects seem to be greater. On the other hand, if of slow growth and of a non-destructive character, a tumor may displace large portions of a hemisphere, especially in the latent regions of the right side, and attain enormous proportions, without producing notable symptoms. This pressure is felt not only in the vicinity of the tumor, but throughout the encephalon, and may cause symptoms and even inflammatory disturbance at a distance. Tumors so located as to obstruct the return circulation from the ventricles by occluding the straight sinus and the veins of Galen produce a dropsical condition of these cavities, an internal hydrocephalus, with extreme pressure indications. Even the bony walls of the cranium over the tumor are thinned and eroded in some instances, and that, too, when the tumor is not superficial. From local pressure conditions interfering with the circulation some edema of the brain in the neighborhood of the tumor is frequently found. In addition, the irritation produced by the tumor in some cases causes an actual proliferation of the glial fibers in the adjacent portions of the brain with decided macular hypertrophy of the convolutions or even of the entire hemisphere. This tendency, according to Spiller, may have much to do with herniation of the brain after decompressive and other operations.

A number of observers have noted changes in the spinal cord in brain-tumor cases. These seem mainly to affect the posterior portions of the cord and the posterior roots. Ursin<sup>1</sup> considers them of toxic character, and Labarsch has found somewhat similar changes in a case of gastric cancer. The analogous changes in anemias and cachexias are significant in this connection. Kirschgasser,<sup>2</sup> however, believes the cord changes to be secondary to increased tension in the dural sheath. Batten and Collier<sup>3</sup> take a similar view, and find such changes in over half of all cases. Certain cases in which the tumor disintegrates the motor tract within the skull show degenerative changes in the direct and crossed pyramidal tracts in the cord.

**Symptoms.**—The symptomatology of brain-tumors presents the widest variations. The clinical picture is dependent upon the situation of the growth and modified in its evolution by the nature of the tumor. A slow-growing mass starting from the meninges may deform an entire hemisphere without giving rise to symptoms, while another of insignificant volume may produce the most marked motor, sensory, and mental disturbances, or lead to sudden death. We may divide the symptoms of brain-tumor into: (1) Those which are presented irrespective

<sup>1</sup> "Deutsch. Zeit. für Nervenzh.," 1892.

<sup>2</sup> "Deutsch. Zeit. für Nervenzh.," Bd. 12.

<sup>3</sup> "Brain," 1898.

of the nature and location of the growth,—*general symptoms*; (2) those dependent upon the situation of the neoplasm,—*focal symptoms*; and (3) *typical symptoms*, those at the superficial site. Under certain conditions the general symptoms have some localizing value, as will be pointed out *verbi*.

**General symptoms** of intracranial growths are: (1) Headache; (2) generalized convulsions; (3) mental impairment; (4) double optic neuritis or optic atrophy; (5) vomiting, and (6) vertigo. Temperature changes above and below the normal, alterations in respiration and pulse rhythm, polyuria, glycosuria, insomnia, delirium, stupor, coma, slow speech, and malnutrition are common to brain-tumor and all other encephalic lesions, especially in their terminal stages. In females anæsthesia is somewhat frequently encountered, especially in tumors located near the hypophysis, in the basilar area, and in the cerebellum.

**Headache.**—In the great majority of cases of intracranial tumor headache is an early and persistent symptom. It has no necessary relation to the location of the tumor, and is usually frontal or occipital, less frequently parietal, or at the vertex. Sometimes the eyeball is the seat of pain. It usually increases with the increase in the tumor's size, and is aggravated by any effect or condition which increases arterial tension and cephalic hyperemia. In character the headache is dull, heavy, persistent, often with great exacerbations; frequently it prevents sleep, and sometimes is of an intensity that becomes intolerable and submerges the patient's intellect. It has even been considered the cause of death. In rare instances a persistent, circumscribed headache has been found to conform to the tumor's location, and, therefore, like all diffuse symptoms, it may exceptionally have a localizing value. In such case there is often local tenderness on percussion, and sometimes local elevation of temperature. In cerebellar growths local pain and pain in the neck, with retraction of the head, are significantly common, but a cerebellar tumor may occasion continuous frontal pain. Pain in the distribution of the fifth cranial pair, or in a single branch, is an indication of local irritation of the nerve that may be confounded with the diffuse headache.

**General Convulsions.**—Over one-half of all cases have general convulsions at some period of the disease. They may be the early and only manifestations of the tumor, and indistinguishable from ordinary epilepsy, for which these patients are not infrequently treated during long periods of time. More often the convulsions are preceded by headaches and focal symptoms. As a rule, Jacksonian fits predominate in the generalized form, either by gradation or alternation. While these commonly conform to the clinical type of the ordinary epileptic seizure, a careful study of them sometimes enables one to detect variations from the type that have some significance. The onset is less violent and abrupt. The clonic stage is likely to be protracted to fifteen minutes, a half hour, or longer; there is less depth to the coma, and the subsequent deep sleep may be much abridged or entirely wanting. They rarely appear with the regularity that is common in ordinary epilepsy. Attacks of the *petit mal* variety are rare in brain-tumor, but



do occur, while epilepsy presenting major attacks is rarely without minor manifestations.

*Mental Impairment.*—The final stage of tumor cases is customarily marked by great lethargy and a stuporous condition that may increase into final coma. In lesser degree this belittling of mental vigor is common in the earlier periods of the disease, with or without convulsions. Often a recurring tendency to stupor is encountered, a sort of sleep drunkenness. When the headache is intense, the patient is likely to seek seclusion, hold his head in his hands, and present a picture of dejected indifference that clearly indicates his mental state. He replies slowly to questions or disregards them utterly. He can not think quickly, and is sluggish in all his mental qualities as well as in his physical attributes. The pulse decreases in strength and rapidity, respiration is slow and shallow, and if suffering ceases the patient sinks into a sort of hibernating sleep. From this apathy, which may be more or less marked, he partially rouses, or he may decline into a more comatose state and die of inanition. The two prominent characteristics of the mental state are apathy and suffering, which may be associated or alternate. So far as mental action can be elicited it is usually unclouded, except in rare periods of delirium. Something of this apathy may be noticed in comparatively recent cases, and, as a rule, brain-tumor patients are little worried over their condition. In some cases the patient presents a silly, joking tendency out of keeping with his manifest ill health and alarming symptoms. Such mental changes are most likely to be well marked, and to appear early in tumors of the frontal lobes. Gennelli<sup>1</sup> reports 77 cases of tumor in the frontal lobes accompanied by mental disturbance and 20 in which morbid psychic phenomena were lacking. In rare cases the mental symptoms closely imitate the formulated insanities, such as mania, melancholia, and even hallucinatory paranoia.<sup>2</sup> Knapp<sup>3</sup> with Dupré leans toward the conclusion that the mental symptoms are due to the two factors of pressure and toxic processes set up in the brain by the new growth through cellular disassimilation.

*Optic neuritis, or papillitis,* occurs in about eighty per cent. of encephalic growths. In very rare instances only one nerve-head, usually on the same side as the tumor, has been found affected. In many cases the condition is more marked in one eye than in the other, and there is some reason to think that the more intense inflammation is also usually on the same side as the lesion. (For further details regarding this lesion of the second cranial nerve reference is made to Part II.) In chronic cases papillitis may suddenly appear, but a chronic papillitis or a very insidious one does not belong to an acute or rapidly developing lesion. The natural result of choked disc is atrophy of the optic nerve, which, therefore, has the same significance in the presence of other tumor indications. It must be borne in mind that central vision may not be greatly disturbed when the discs are distinctly choked, and their examination should never be neglected. This general symptom of brain-tumor is very constantly found in growths involving the cere-

<sup>1</sup> "El Psiquiatra," July 15, 1907.

<sup>2</sup> Beyerthal, "Mensch. und Wochens," 1899.

<sup>3</sup> "Brain," 1906, p. 33.

bellum, geniculate bodies, corpora quadrigemina and the brain-axis. It, therefore, has a slight localizing value. Paton<sup>1</sup> has investigated the relation of the location of brain-tumors to optic neuritis in 200 cases, and concludes that primary atrophy is caused by direct or indirect pressure upon the optic nerve or chiasm; that cortical tumors produce choked disc usually, and with a severity proportioned to the proximity of the growth to the chiasm; that tumors affecting the thalamus, mid-brain, and cerebellum are practically always attended by choked disc; that tumors in the white matter and in the pons do not cause choked disc until the cortex or the cerebellum respectively are involved. Cushing and Bodley insist that interlacing and overlapping of the visual color-fields similar to those found in hysteria, are of such common occurrence as to almost justify their being ranked as one of the cardinal general symptoms of brain-tumor. Frequently this disturbance of the color-fields promptly subsides after decompressive operations.

**Vomiting.**—Attacks of vomiting, usually of the so-called cerebral variety, without fermentation, nausea, and effort, are common. They may last for a few days or weeks; subside and recur, and they usually attend other indications of tumor extension or invasion. Vomiting sometimes threatens death by inanition. Like choked disc, it is most frequent in tumors involving the cerebellum, especially the middle lobe and the corpora quadrigemina.

**Vertigo** occurs with brain-tumors in all locations, but especially in the cerebellar and frontal regions.<sup>2</sup> It is a vague distress in some cases or a feeling of being submerged or of mere darkness and sinking, and may be paroxysmal or constant. In addition, brain-tumor may give rise to ocular vertigo by involvement of the motor oculi nerves, and to auditory vertigo by implicating the eighth nerve. In cerebellar tumors affecting its position, especially the middle one, there may be forced movements and attitudes. In these combinations vertigo furnishes a valuable localizing indication. In some cases it is an epileptic equivalent. It sometimes is attended by vomiting. It may prevent the patient's rising from recumbency, or cause him to lie down if standing. Ordinarily he grasps an object for support, and is soon over the attack.

**Vocal Symptoms.**—The symptoms due to the presence of a new growth or other lesion in special brain regions have been discussed at length in the opening chapters of this part. They consist usually of unilateral *aparesis* confined to the face, to a limb, or to a segment of a limb; of *acroparesis*; of *parasthesia* of functional distribution; of *hemianopsia* or other sensory disturbances; of *aphasia* and *aprosia*; of *stereognosis* and of impairment of the *cranial nerves*. They are variously combined as associated anatomical structures happen to be involved. They advance with the tumor's growth, and change as irritation yields to destruction. They thus vary indefinitely in different cases, and usually present numerous modifications in the clinical history of any given case. Williamson<sup>3</sup> gives particular importance to the significance

<sup>1</sup> "Brain," 1900, p. 45.

<sup>2</sup> "Brain," *Wien. Klin. Woch.*, 1897, No. 49.

<sup>3</sup> "Practitioner," Sept., 1904.



of hemiplegia of very gradual onset which may at first be entirely unattended by the other usual symptoms of brain-tumor. To the Jacksonian fit, whether the spasmodic or the sensory features predominate, most attention has been directed, as it points with definiteness to the locus of greatest and usually of initial disturbance. To avoid needless repetition, the reader is again referred to the previous chapters on cerebral localization and diseases of the cranial nerves. *Hallucinations* of the special senses occurring with convulsions or independently sometimes are caused by tumors situated in the corresponding sensory cortical area or influencing it more or less directly.

**Topical Symptoms.**—The location of a new growth near the surface of the brain, so that it impinges upon the meninges or invades them, is sometimes attended by topical conditions of some significance. *Local and persisted tenderness, heat, and headache* may be thus induced. In the rare cases, where the cranial bones are perforated by erosion, *protection* may detect the bony opening and the tumor mass. Invasions of the orbit, pharynx, and nasal cavities are open to ready investigation. *Percussion* over large tumors superficially placed may demonstrate a changed pitch in the resulting note.<sup>4</sup>

*Multiple tumors* may furnish many diverse and conflicting symptoms, but usually one, owing either to its situation or major size, gives a preponderance of manifestations.

**Course.**—The majority of cases of brain-tumor are clinically of insidious onset, progressive development, and final termination in a cachectic state, induced by the gradually increasing intracranial pressure and the extinction of cerebral functions. The ordinary duration is from a few months to three years. The lethargy, stupor, and coma of late stages are frequently aggravated by paralytic features, and continuous vomiting may defeat all attempts at nourishment. While this is true of the larger number of cases, there are numerous exceptions. Sometimes the first pronounced symptom is an apoplectic attack which may carry off the patient at once. The apoplexy is brought about by a cerebral hemorrhage, due to erosion of a vessel, or sometimes to the tumor pressure obliterating an artery by thrombosis or by its mere mechanical effect. In the softer growths, notable gliomata, hemorrhage into the mass may take place and secondarily affect the cerebral structure. Hemorrhage into the substance of a tumor may also directly induce the apoplectic state and lead to a fatal termination. Gerhardt states that in this accident the initial fall of temperature that attends an ordinary cerebral hemorrhage does not occur. Some tumors give rise to no symptoms and are only detected after death from intercurrent disease.

The nature of the growth in some degree determines the rapidity of the evolution of the case, but in turn is subject to its localization. Tubercle, fibroma, osteoma, sarcoma, syphiloma, carcinoma, and glioma, in ascending order, increase in the rapidity with which they influence the brainstructure. The more rapid the growth, the more rapid the development of pressure and the more quickly does destruction advance. Tubercles, however, furnish some of the most rapid as

(Beauz, "Wien. Klin. Wochs.," 1887, No. 48.)

well as some of the most dilatory cases. A fatality is imminent in proportion as the growth recedes from the cortex and approaches the medulla in location. In the latter position or in its neighborhood, as in the basal ganglia, the pons and cerebellum, a small and comparatively benign growth may induce a rapid course and early fatal termination.

The succession of symptoms is again determined by the nature and the location of the growth. Headache is, of all the diffuse or general symptoms, commonly the earliest, and optic neuritis usually follows in a few months. Spasms depend on the amount of irritation of the cortical structures, and paralysis on their destruction or the cutting of the motor paths. General convulsions, as pointed out, are often early features; they especially precede the paresthesias and palsies. By the extension of the growth invasion symptoms are set up (see p. 192), and a widening of motor and paralytic phenomena follows in anatomical order. Tubercular and carcinomatous growths, being usually secondary manifestations, bear with them the possibility of a rapid downward course from conditions outside the cranium. In addition, a tubercular necrosis is likely to set up a diffuse tubercular meningeal infection that may promptly destroy the patient. A syphiloma in the same way may be attended by a wide-spread syphilitic meningitis.

**Diagnosis.**—In a case presenting cerebral indications and giving rise to the suspicion of tumor, the diagnosis is much facilitated by a careful arrangement of the symptoms in the order of their development. A number of problems are presented: (1) Is there a tumor? (2) Where is it located? (3) What is its size? (4) What is its nature? (5) Is it operable?

To the solution of the first question, is there a tumor, a definite answer can usually be given. Bruns says tumor may be diagnosed in eighty per cent. of all cases. After taking into consideration the evolution of the case, the focal or localizing symptoms are the most reliable data, but are strongly confirmed or rendered positive by the presence of the diffuse indications. Localized fits may be presented by ordinary epilepsy, but if headache, cerebral vomiting, vertigo, and choked disc, one or all, are added, the probability of tumor is enhanced. Should now the focal spasm be followed by persistent paresis or paresthesia in the same locality, a positive statement is allowable. In the same way a hemianopsia or aphasia, following or associated with generalized or limited convulsions and attended by some or all of the diffuse symptoms, permits a diagnosis of tumor.

The clinical history of the case is important, as by it and its special symptoms we have to differentiate tumor from acute and chronic meningitis, with which, indeed, it may in some cases be complicated. The early and persistent headache is common to both, but tumor presents, as a rule, a choked disc, while meningitis has a neuroretinitis. Meningitis, even of the tubercular form, is of comparatively rapid development, while tumor commonly requires months. In meningitis the motor symptoms are usually bilateral, as contrasted with the one-sidedness of tumor disturbance. It will be recalled that a latent cerebral abscess may present all the indications of a tumor, which indeed it is, and it



requires no differentiation aside from the fact that it offers a better operative outlook. The *cerebral palsies of childhood and general paresis* in adults have been confounded with tumor, but a careful scrutiny of the case, with a knowledge and mindfulness of these analogies, will obviate error. The tumors which present a sudden apoplectic onset may be confounded with *cerebral hemorrhage or softening*. Here the ordinary antecedents of vascular disease and the arterial condition presented furnish a strong presumption of vascular accident, and serve to a reasonable degree to exclude tumor, if the diffuse symptoms of tumor are lacking. It will, however, be borne in mind that a brain-tumor may bring about vascular disease, and that hemorrhage into gliomata and other soft growths may produce apoplectic states which would be further favored by atherosclerotic conditions of the blood-vessels. The diagnosis must rest on the antecedent and sometimes on the subsequent history of the case. Some cases of paralytic angina may be difficult to differentiate. The headache and vomiting may be attended by temporary blindness, aphasia, local numbness or actual paresis of the extremities on one side, and mental peculiarities varying from hebeticude to actual delirium. The paroxysmal features of such attacks with full relief in the intervals and the usual history of heredity to migraine help the diagnosis.

There is reason to hope that skiagraphy may give aid in the diagnosis of brain tumors. Ottéi and Balliè<sup>1</sup> were able to thus demonstrate a sarcoma in a calvaria. The writer,<sup>2</sup> Mills,<sup>3</sup> and many others have obtained similar results during life. Great vascularity, hemorrhage into the tumor, or calcareous changes in or about the mass favor the x-ray detection of the growth.

In Europe Nonne, Oppenheim, Grasset, in this country Hoppe and others have called attention to numerous cases of *pseudocerebral tumor* presenting many of the most important symptoms of tumor, but either ending in recovery or showing no pathological changes at operation or necropsy. Finkelsburg and Eschmann<sup>4</sup> also report a number of such cases, but find a chronic leptomeningitis or hydrocephalus with neuritis to be present.

In *verruca serena*, particularly in chlorosis, headache, vomiting, malnutrition, syncopic attacks, and even choked disk may furnish a very great resemblance to the manifestations of brain tumor.

The second question, of *location*, is answered, if answerable at all, by a consideration of the focal and topical symptoms of the case and the localizing bearing of the diffuse symptoms that are presented. The absence of focal symptoms points to the regions of latent lesions in the frontal, temporo-sphenoidal, and postparietal regions, especially on the right side.

It is necessary to answer the second question before the third can be approached, as we can only relatively estimate the size of a tumor by knowing its location and the anatomical regions invaded. To this end the sequence of developments is our greatest aid. Invasion symptoms

<sup>1</sup>"*Rivista di Patologia*," Oct., 1902. <sup>2</sup>"*Amer. Jour. Med. Sciences*," Feb., 1890.

<sup>3</sup>"*Pitts. Med. Jour.*," Feb. 23, 1902. <sup>4</sup>"*Deutsch. Zeit. für Nervenhe.*," 1900.

enable us to trace, especially in the motor cortex, the gradual growth of a tumor, and in some directions to indicate its extent. If this can be done on a portion of the periphery, a knowledge of the symptoms that would arise by the tumor's extension in the opposite direction may enable us to say whether or not such centers or pathways have been seriously invaded. It is evident that only a very inexact measurement can be made, and this is especially true if the growth is situated in the neighborhood of the latent lesion territories.

To the fourth question, what is the nature of the tumor, a positive answer can never be given, unless there is an external portion of the tumor, but a strong probability is frequently forthcoming. In this relation the age, the diathesis, the history of previous illness, the presence of various diseases, the location of the tumor, its rate of development, and the effect of treatment aid us. In childhood, tubercle, glioma, sarcoma are most frequent; in adults, the firmer tumors and syphiloma; in advanced years, carcinomata. The presence elsewhere of tuberculosis, carcinomata, parasitic cysts, or syphilis gives much weight to the supposition that the brain-lesion is of a similar character. Tubercle and glioma favor the pons and cerebellum. Syphilis favors the pons, basilar area, and cortex, but is rare in the cerebellar white matter and the vertrum ovale. Fibroma and glioma, being interstitial growths, occur in the deep structures. Sarcoma mostly occurs in the ventricles or meninges.

A rapid onset, followed by a stationary period, speaks for tubercle, especially in the first half of life. Glioma and sarcoma are of insidious and steady development. Apoplectic seizures in tumor cases usually mean glioma. Syphilis provides a rapid onset and course.

Only syphilitic tumors are permanently affected for good by treatment, but it must be said with due emphasis that the iodids are capable of apparently checking circumscript growths and frequently cause temporary and misleading benefit in all forms of tumor, probably by forcing the removal of the circumscribing edema.

The fifth question concerns the possibility of surgical removal. From the large statistics compiled by Starr, and from later additional data, it can be stated that not more than seven times in a hundred cases of brain-tumor is the growth amenable. Unless the neoplasm lies on or in the convexity of the cerebral hemispheres it is not readily approachable. Piller<sup>1</sup> tabulates forty-eight cases of cerebellar tumor surgically removed. In twenty death promptly ensued; in sixteen improvement with subsequent death from recurrence of the growth; in four almost complete cure, and apparently complete cure in one only. Frazer,<sup>2</sup> from a study of 116 operations of recent date, draws the following data: Recovery, 16 per cent.; improved, 28 per cent.; unimproved, 15 per cent.; mortality, 42 per cent. Cerebellar cysts offer a good operative prospect. The basilar, pontine, and medullary regions are out of the operative field excepting growths in the cerebellopontine angles, which usually are of extra-cerebral origin, and are comparatively favorable for surgical attack. Cysts and old abscesses are readily drained. Sarcomata can usually be enucleated, tubercles and fibroid tumors can be shelled

<sup>1</sup>Arch. Pract. & Chirurg. 1901.      <sup>2</sup>N. Y. Med. Jour., Feb. 18, 1906.



out, but glioma, from its infiltrating character, and other similar growths can never be entirely removed, and grow again if the attempt is made.

**Prognosis.**—If ninety-three per cent. of brain-tumors are inoperable, and syphiloma is only partially amenable to medicinal treatment, the gravity of the disease is apparent. The great majority of cases run their course within three years, though slow-growing neoplasms may exist almost an indefinite time or may only furnish a post-mortem surprise. The outlook turns upon the nature of the growth and the associated clinical manifestations. The possibility of sudden death should not be overlooked, especially in tumors located in or near the brain-axis.

**Treatment.**—The management of brain-tumors is of two sorts: (1) That directed to the tumor itself, and (2) that to the general physical condition. Removal of the tumor by operation is possible in a small number of cases, and should be done whenever indicated and the conditions are otherwise favorable. This surgical proceeding in the hands of competent men has secured some brilliant results and saved and prolonged life. Oppenheim states that good results have been obtained in about one-half of all well-selected cases. Even in inoperable tumors a wide opening of the skull has relieved pressure, has benefited the mental condition, stopped the headache, and caused the choked disc to subside. It is indicated in at least five-sixths of all cases, according to Knappe.<sup>1</sup> Indeed, in some cases it has seemed to cause the tumor to recede. The simple operation of Anton, puncture of the corpus callosum, seems to produce a permanent drainage of the ventricles, and in tumors of the base has proved a valuable procedure. As above indicated, cerebellar tumors are not the most favorable for operation. The harder and slower growing sorts of brain neoplasms offer the best operative results. Tubercle, sarcoma and fibroma, or their varieties, can be removed en masse, while the limits of infiltrating growths are difficult to distinguish and complete removal is practically impossible. Cysts and abscesses can be evacuated, and if the secreting wall is removed, a practical cure follows. Should the cyst be due to degeneration of a sarcomatous growth, the probability of a return is very great.

In syphilitic growths intensive treatment with mercury, arsenic, and the iodids usually produces prompt improvement. This goes so far that many cases are alleged to be cured, and the shriveled remains of a syphiloma have been found post-mortem to testify to the efficacy of treatment and the precision of diagnosis. A large degree of reservation, however, should be maintained in every syphilitic case. As a rule, the cure is not complete. Some residuum of disability can usually be detected, and a constant tendency for the syphilitic process to reappear during the rest of life too frequently keeps these patients fighting the disease as long as they live. In order to secure the best results mercury, arsenic, and iodid should be used, either together or in succession, depending upon the urgency of the condition, and, carefully guarded, should be pushed to the limit of toleration. Explicit directions for the treatment of syphilis will be found in the chapter on Syphilitic Diseases of the Nervous System, Part VI.

<sup>1</sup> *Boston Med. and Surg. Jour.*, "Oct., 1890.

As tumors of all varieties have shown at least temporary improvement under the use of anti-syphilitics, the practitioner must be guarded in drawing inferences from such therapeutics, and not allow himself to take too favorable a view of the case when this occurs. It is an established rule to use specific treatment in all cases of brain-tumor where there is a shadow of a doubt as to their character. If, after three weeks of vigorous treatment, no benefit is obtained, the lesion is pretty surely not syphilitic. Should improvement take place, a continuance of treatment should be persisted in until all symptoms have practically disappeared. A return of former symptoms or further development of tumor indicates in the face of specific treatment practically demonstrates the non-syphilitic character of the disease. Specific treatment must never be depended upon if blindness is threatened. A decompressive operation should always be urged if the choked disc be intense, or if a notable and progressive narrowing of the visual field or sclerotic changes about the vessels of the disc indicate a beginning atrophy. Horsley contends with much reason that when attackable, even syphilomata should be treated surgically after a fair trial of specific medication, and Owens Knapp<sup>3</sup> found nine out of ten such operations to have been successful.

The patient's general condition will require constant attention. Tuberculous and other cachectic states have their own requirements. The headache can often be relieved for a time by brisk cathartics and hot baths. Antipyrin and other coal-tar analgesics often control the headache for a time. The opiates are frequently powerless except in extreme doses, and their use should be postponed to the last possible moment. Vomiting yields best to nerve sedatives, such as the bromide, and to measures like hot baths and mustard foot-baths, calculated to decompress the cerebral circulation. In rare cases it is quite unmanageable, even by morphia, and may lead to rapid inanition and death from exhaustion. Convulsions can usually be restrained by the bromide. The continued use of bromide and antipyrin will be found especially valuable in these cases, and a flagging heart may be protected by caffeine or strychnin. The optic neuritis can be benefited in syphilitic cases by medicinal treatment. In other sorts of growth it is frequently benefited by operation. Repeated spinal puncture may also yield palliative results. It must be used with caution, as in cases of great pressure, and particularly when the growth is in the posterior fossa, the reduction of intracranial tension may lead to the downward forcing of the brain at the foramen magnum, causing medullary pressure and serious symptoms or even death. When optic atrophy has once occurred, it is permanent. Finally, by nutritious diet, baths, massage, and general measures the strength of the patient is supported and life prolonged.

<sup>3</sup> *Boston Med. and Surg. Jour.*, Oct., 1899.



## CHAPTER XII.

## HYDROCEPHALUS.

**HYDROCEPHALUS** is a term loosely used to designate any undue amount of watery fluid within the skull, and in such a sense is synonymous with dropsy of the brain. Tubercular meningitis, frequently called *acute hydrocephalus* by older writers, is not the condition in question. Nor are we now to consider the compensatory increase of cerebrospinal fluid occurring in the convolutional shrinkage of old age, or in the cerebral atrophy of dementia, or in postencephalic or anencephalic defects. The question does not pertain to the increase of fluid which marks acute, serous or tubercular meningitis, or to the edematous state that frequently attends cerebral tumors and cerebritis. Attention has also been called in the proper place to ventricular distention resulting from tumors situated in the posterior cranial fossæ, which mechanically block the interventricular passages, especially that between the third and fourth ventricles.

It is desired to restrict the term *hydrocephalus* to a congenital or acquired, acute or chronic condition marked by great increase in the amount of cerebrospinal fluid within the skull attended by compression of the brain. In some cases it is entirely ventricular, constituting *internal hydrocephalus*; in others it is subdural, constituting *external hydrocephalus*. In every instance there is a disproportion between the secretive activity of the choroid plexuses and the absorptive function of the Pacchianian bodies and the venous outlets.<sup>1</sup>

**Etiology.**—The causation of hydrocephalus is obscure. Some families seem to be marked by a hereditary tendency to it, as shown by several cases occurring in the same or succeeding generations. Hereditary syphilis has for long been thought to be a competent cause, but in many cases it can be excluded with a reasonable certainty. Its causal relation is supported by Heller<sup>2</sup> and by Timmanko<sup>3</sup> in careful studies. Alcoholism on the part of the parents is also supposed to have some part in producing the congenital variety, and is doubtless active in some of the late adult cases. Traumatism after birth has produced it, and it has been noted as a sequel of cerebrospinal meningitis<sup>4</sup> and of Quincke's *acute serous meningitis*, the symptoms of which do not vary materially from those of the ordinary infectious variety, but it is marked by the rapid production of subdural hydrocephalus and great intracranial pressure.

A *pituitary tumor*, a tumor in the neighborhood of the pituitary gland, or in the pontocerebellar region is sometimes found.

Frazier<sup>5</sup> suggests four forms: (a) the obstructive, (b) the nonabsorptive, (c) the hypersecretive, and (d) the occult.

<sup>1</sup> Browning, "Med. Record," May 27, 1916.

<sup>2</sup> "Deut. med. Wochensh.," June 20, 1890.

<sup>3</sup> Trans. Section on Diseases of Children, International Med. Cong., Rome, 1904.

<sup>4</sup> E. P. Joslin, "Amer. Jour. Med. Sci.," Oct., 1909, eight cases.

<sup>5</sup> "Amer. Jour. Dis. of Children," Feb., 1906.

**Morbid Anatomy.** In congenital cases and in those occurring before the cranial bones are firmly united the head is enlarged, sometimes to vast proportions. The cranial bones are usually reduced in thickness, often to that of paper, the diploe being absent. At the same time they are frequently much broadened. The sutures are patent, or supernumerary bones are commonly found if synostosis has taken place after the disease has been present for some time. The frontal, occipital, and squamous portions of the temporal bones are displaced outward. The parietals conform to the globular shape of the head.

The amount of fluid may be incredibly increased, and as much as three gallons has been noted in a very extreme case of long standing. It is a colorless fluid of low specific gravity, quite similar to normal cerebrospinal fluid.

The principal distention is usually in the lateral ventricles, which bulge in all directions and stretch out their cerebral walls into a thin lining for the enlarged cranial cavity. The basal ganglia are often compressed and flattened. The ventricular lining is found usually, if not always, in a thickened, granular, hypertrophic condition. It may reach a thickness of half an inch. The choroid plexuses are correspondingly enlarged. This apparent ependymitis often serves to occlude the ventricular aqueducts, so that the third and fourth ventricles may not share in the dilatation or it may be confined to one lateral cavity. In about one-half of the cases connection with the spinal spaces is obliterated. In the congenital and adult cases it is common for all the cerebral ventricles and the cord to be involved. When the third and fourth ventricles are affected, the optic tracts suffer and optic atrophy is common. At the same time the cerebellum, pons, and medulla are defectively developed. The meninges may show little or no change. After cranial solidification the anatomical conditions are modified by the resistant character of the skull. The amount of fluid is necessarily less, but the pressure falls more directly upon the cerebrum.

**Symptoms.**—Hydrocephalic enlargement of the head sometimes renders birth difficult or requires the use of the perforator before extraction can be accomplished. In other and a majority of the cases the condition is insignificant or unnoticed at birth, and appears during the first year of life, especially during the first six months. The head gradually or rapidly enlarges in all its diameters and measurements. An increase in the circumference of the head at a rate of a centimeter daily has been noted, but usually it requires several weeks for that amount of enlargement to take place. The outline of the skull is globular and may overhang the face, ears, and occiput, which are not correspondingly enlarged. The internal pressure is manifest at the bulging fontanelles, which are increased in size and connected by wide-open sutures. The return circulation of the cerebrum is impeded, and the collateral veins in the scalp and face become distended and strikingly apparent. From the outward tilting of the frontal bones the orbits are directed downward and the ocular globes are often maintained in the same direction, even to such a degree that the cornea is only seen with difficulty. Optic atrophy and



blindness are frequent. *Nystagmus* and *strabismus* are common. *Flaccidity* is readily obtained on palpation, and the head, in extreme cases, may be translucent.

The hydrocephalic child shows little activity, can not raise its head sometimes, or only does so with the aid of its hands. There is a tendency to peevishness and restlessness or somnolence and coma, broken by generalized convulsions and a frequently repeated distressed cry. Older children complain of pain in the head. The body and limbs suffer in their nutrition and are unequal to the task of holding up the head and trunk. In some cases *spasticity* develops in the limbs, especially in the lower ones, and some paralytic loss of power is frequently noted. Feeding is frequent and may be provoked by movements or much handling.

If not rapidly fatal by exhaustion, convulsion, coma, or syncope, the disease may come to a standstill or proceed with such slowness that the child is enabled, in some defective measure, to maintain growth and develop its physical and mental faculties. These are both commonly much retarded, so that the body is dwarfish and in great disproportion to the cephalic enlargement. The occasional cases that live to mature years are more or less imbecile, clumsy, and physically defective, though a slight degree of hydrocephalus is not incompatible with mental brilliancy. Many cases of marked dolichocephalus in adults with beetling brow and salient occiput prove the possibility of recovery.

When hydrocephalus is acquired subsequent to cranial synostosis, the symptoms are vague, and only rarely can the condition be deciphered ante mortem. The indications are not unlike those of tumor. It usually follows head injury. Mental impairment, especially of memory, vertigo, vomiting, insomnia, headache, convulsions, and rigidities are encountered. Hemiplegia is rather frequent. The pupils are dilated and stationary. Strabismus is common. Periods of coma are frequent. Death may be sudden or follow coma.

**Course.**—The disease presents a varied course. The congenital cases may run rapidly to a fatal termination in a few weeks, while others come to a standstill and allow a fair degree of adult development and a comparatively long life. As a rule, marked hydrocephalic cases do not reach maturity, but give out at adolescence and puberty, if not sooner carried off by convulsions or comatose conditions referable to the intracranial pressure. It is extremely rare for them to live beyond thirty. In the acquired adult cases a fatal termination is usual within two or three years, and often sooner.

**Diagnosis.**—The diagnosis in infantile cases can hardly offer any difficulty if the tendency to cephalic enlargement is noted. The globular shape of the head should distinguish it from the rectangular conforma-



Fig. 306.—Chronic hydrocephalus in a child of four years. Circumference of head, 25 inches.

tion of rickets with the enlarged and squared forehead and prominent frontal eminences, though patency of fontanelles is usually present in both. The appearance of rickety conditions in the long bones and at the costal extremities is also significant. The two conditions, however, may be associated. Before the head shows much or any abnormal increase it is difficult to exclude meningitis, which may, indeed, be the causal condition.

Dandy and Blackfan<sup>1</sup> have found that occlusion of the ventricular outlets can be determined by the injection of phenolbiphenylthalein into the ventricular cavity, where it is retained, only very faintly appearing in the urine and does not appear at all in the spinal fluid. At the same time if injected into the cerebral meningeal spaces it appears in the spinal fluid in six to eight minutes, as it would in the normal case, whether this injection were practised intraventricularly or into the pial spaces. In a similar way occlusion or patency of the passages between cranial and spinal pial spaces may be determined.

In adults the diagnosis is well-nigh impossible, and when suspected can only be confirmed by an autopsy. A condition acquired after complete union of the cranial bones, commencing anywhere from five to fifty years of age,—hyperostosis cranii,—may at first sight mislead. The history will at once differentiate it. Notable hydrocephalic enlargement must begin in very early life. In hyperostosis cranii the size of the head is due to a thickening of the cranial bones, and the bones of the face and spine are usually affected in a similar way. The enlarged head of acromegalia may be distinguished by its late development and the associated deformities of face, hands, and feet.

**Prognosis.**—The prognosis for life is always grave. The great majority of cases die within a year. A few with enormous heads live for a few years, and in those marked by a stationary condition life is still usually much shortened. The outlook for mentality is also darkened, but must be estimated for the individual case. Mental enfeeblement is the rule, and this may be mere childishness or pronounced imbecility. Epileptoid attacks are of serious import both as to life and mental development.

**Treatment.**—The treatment of these cases is medical and surgical. Mercurialunctions to the head and the use of iodine preparations have long been practised. It is probable that some of the good results attributed to these measures are not due to their alleged stimulation of absorption, but to their influence on a syphilitic factor. As a rule, they are of little value, and should only be used when there is suspicion of specific taint. Catharsis and other violent elimination is to be discontinued, as it only serves to exhaust the patient's diminished strength. When the process is active, the application of cold to the neck and head by ice-bags or flexible coils is valuable. A slight mercurial action, preferably by calomel in minute and frequent doses, should be used. Convulsions require sedatives, especially bromids.

Strapping the head with surgeons' plaster or the application of elastic caps and bandages have been advocated, but are usually intolerably pain-

<sup>1</sup>"*Ann. Amer. Med. Assoc.*," Dec., 1913, p. 2214.



ful and aggravate the pressure conditions. Repeated tapplings through the fontanel, or by lumbar puncture have been employed with varying results. Occasionally they have seemed to be successful, but lumbar puncture will be fruitless in at least half of the cases owing to the lack of communication between the brain and cord spaces.<sup>1</sup> Only a moderate quantity of fluid should be withdrawn at a time, and the strictest antiseptic precautions must be employed. Some have ventured to withdraw fluid and inject iodine solutions as in the treatment of hydrocele, but the plan can not be advocated. Too frequently the punctures result in a meningitis that carries off the patient. Plans of constant external drainage have been devised, and, while attended in some cases with temporary benefit, the result has been uniformly fatal.

A case has been reported by Rokitsansky in which spontaneous rupture under the scalp led to a recovery. The idea occurred to Dr. L. L. McArthur, of Chicago, to drain the ventricular cavities into the areolar spaces beneath the scalp by the insertion of drainage-tubes or silk into the cranial cavity through a drill-opening above and back of the ear. He attached the drainage material to the pericranium, and then, securing first-intention healing in his overlying scalp-flap, allowed the serous accumulation to flow. A hygromatous swelling forms under the scalp, from which absorption seems to be rapid, and the head diminishes in size if the bony sutures are not united. This operation was done for the writer on the case shown on page 273, with immediate and lasting improvement. In two other cases it also demonstrated its utility. Mikulicz and Troje<sup>2</sup> have had favorable results with the same method independently devised. The insertion of a drainage-tube into the ventricle and terminating in a broad flange under the dura to which it is secured, thereby connecting ventricular and meningeal areas, is probably the best plan, unless the simple method of puncture of the corpus callosum, devised by Anton ("Balkenstich") proves better. Others have established drainage from the lumbar spinal spaces into the peritoneum. Even the fourth ventricle has been opened by the surgeons and the choroid structures clipped off with distinct advantage in the cases that survived, probably due to the establishment of permanent drainage. The disadvantage of all surgical procedures is the liability of infection and meningitis, and the peculiar tendency of these cases to sudden death from removal of fluid or from the inhibition of the heat-controlling mechanism, which allows the temperature to exhaust the patient in a few hours. Frazier<sup>3</sup> contends that thyroids act to inhibit the glandular activity of the choroids. In the non-absorptive and hypersecretive cases systematic thyroid feeding should be given a persistent trial.

<sup>1</sup>cf. Astruc, "Les Hydrocephales," Paris, 1806.

<sup>2</sup>"Centralblatt f. Chir.," Sept. 5, 1899.   <sup>3</sup>Loc. cit.

## PART IV.

# DISEASES OF THE SPINAL MENINGES AND SPINAL NERVES.

## CHAPTER I.

### SPINAL MENINGITIS AND SPINAL MENINGEAL HEMORRHAGE.

SPINAL meningitis is an inflammation of the covering membranes of the spinal cord. The varieties of meningitis ordinarily described have been somewhat arbitrarily based upon their anatomical location. The terms *pachymeningitis* and *leptomeningitis* are respectively employed, as the dura or the softer membranes are principally involved, but a sharp division is clinically impossible, and is not found post mortem. For purposes of description we may consider: (1) *Pachymeningitis*, or external and internal inflammation of the dura; and (2) *leptomeningitis*, or inflammation of the pia. Inflammation of the inner surface of the dura, from contiguity, must involve the leptomeninges more or less, so that the conditions are usually associated, and meningitis, originally external, very readily invade the pia. Owing to the very intimate relation of the pia and the fibrous septa of the cord, association with myelitis is frequent. A mixed form, *meningo-myelitis*, is common.

**Pachymeningitis Externa Spinalis.**—*Pachymeningitis externa*, or external dural meningitis of the spine is due to chronic irritation and inflammatory conditions invading the spinal canal, and is, therefore, secondary to other morbid states. Vertebral tuberculosis, Pott's disease, abscesses and new growths near the spine, inflammation and purulent collections in the pleura, mediastinum, peritoneum, and pelvis, and sacral bedsores may be the source of the meningeal thickening. This gives rise to symptoms mainly by irritation of the sensory and motor nerve-roots passing through the area of disease. When the thickening becomes extreme, as occasionally happens, it may be sufficient to compress the cord and give rise to pressure symptoms and a specific paraplegia similar to that of a cross-myelitis. The condition is usually due more to the inflammatory invasion of the cord than merely to pressure. There is local tenderness over the spine, shooting or constant pains in the distribution of the irritated nerves, twitching of their muscles, and hyperæsthesia in their cutaneous areas, which may go on to anesthesia and



muscular palsy if the nerves be sufficiently compressed or inflamed to cause their degeneration.

Anatomically, the dura is found hyperplastically thickened, with much adventitious fibrous tissue, and is frequently covered by a caseous or purulent deposit or involved in a new growth. The various findings, of course, depend upon the nature of the primary disease. When the thickening is extreme, the soft membranes are adherent to the dural tunicence and may be indistinguishable. The cord then shows a constriction, and may, in severe cases of long standing, be very considerably reduced in size at the place of disease presenting local myelitis and secondary ascending and descending degenerations.

The diagnosis is usually not difficult if the primary disease is recognized. It may be confounded with myelitis, with which it is often associated late in the case. The clinical history shows a preponderance of pain, spasm, and irritation, a chronic course, and an early absence of paralysis; while in myelitis the rapid onset, the relative absence of pain, aside from the girdling sensation, and the promptly developed paralytic state with early bladder and bowel symptoms are distinctive.

Owing to the serious nature of the causal conditions, the prognosis is bad and treatment is practically surgical. The pachymeningitis externa associated with Pott's disease is perhaps the least grave, as the proper orthopedic and surgical management of such cases frequently is followed by practical recovery even when the cord has probably been notably compressed.

**Pachymeningitis Interna Spinalis.**—Pachymeningitis interna, or internal inflammation of the spinal dura, is described as (1) hypertrophic and (2) hemorrhagic. In reality, these forms are but stages of one and the same process, the thickening and hypertrophy following upon the organization of the hemorrhagic exudate. The term "hematoma of the spinal dura mater" has been sometimes used. The condition is a rare one, and usually the cerebral meninges are also similarly affected. It is most commonly found in general paralysis of the insane and chronic alcoholism.

The portion of affected dura presents on its inner surface a very considerable thickening, which may be a layer of reddish-brown exudate or consist of laminations of fibrous tissue, the apparent result of the organization of successive hemorrhagic exudations. It may attain sufficient size to constrict the cord. The softer, more recent, and reddish or brownish layers consist of fibrin and blood. The distribution is frequently extensive, but in some instances it is confined to a comparatively short vertical extent of the envelope of the spinal cord, and is then more frequently situated in the cervical region. This circumscribed cervical form was first described by Charcot and Joffroy, who give it the name of *pachymeningitis cervicalis hypertrophica*.

Syphilis, trauma, alcoholism, and exposure are regarded as competent causes, and hence it occurs, as a rule, in adult males, though some cases in children are reported.

The condition is essentially chronic and of slow onset. At first irritation of nerve-roots gives rise to local pain and hyperesthesia over the spine and in the peripheral distribution of the spinal nerves of

corresponding origin. This is followed, months or years later, by gradual loss of power, atrophy, and anesthesia in the corresponding parts, and as compression upon the cord is produced spastic symptoms appear below, with increased reflexes, rigidity, and paraplegia, leading sometimes to exhaustion and death. Some cases present stationary periods, and a few recoveries are claimed. The muscles of the forearm are not uniformly affected, the flexors being most impaired. This results in a peculiar deformity that is striking and almost characteristic. The small muscles of the hand usually suffer and both arms are commonly affected, though not usually in equal degree. In many instances the cord shows the peculiar changes of *syngomyelia*, and the symptomatology of this condition is then added to that of the pachymeningitis.

The *diagnosis* is difficult when the dural involvement is of general distribution and cerebral symptoms are present, as the spinal features are overshadowed. Diseases of the spine, progressive muscular atrophy, craniomyelitis, tumor, and external pachymeningitis must be excluded. An operation may be required to differentiate the external dural inflammation. It presents, except in syphilitic cases, the best chance of favorably influencing the condition and preventing destruction of the cord. In the



Fig. 331.—Position of hand in pachymeningitis cerevralis hypertrophica.

desperate situation that is presented and with the courage given by asepsis, it may the more reasonably be resorted to early. Where syphilis is suspected, specific treatment should be persistently tried.

**Acute Spinal Leptomeningitis.**—Acute spinal leptomeningitis, or inflammation of the pia mater, is due to infection. It usually involves the inner surface of the dura, and commonly extends to the peripheral substance of the cord.

**Etiology.**—The infection of cerebrospinal meningitis, as in epidemics of the disease, fills sometimes only on the cord, and the infective nature of the attack is obvious. In those cases, however, that are attributed to exposure, insolation, rheumatism, and other occult conditions, the infection is less readily comprehended, but in all probability is equally in operation, being favored by the physical conditions mentioned. In some recent cases the discovery of the pneumococcus and the meningococcus proves the identity of the infection with that of the cerebrospinal type, to which the reader is referred. The association of cases with septicemia, pyemia, and other infectious blood-states points again to infection, and in the



lymph and spinal fluid of these parts abundant pathogenic organisms have been observed. In some instances the spinal disease is an extension from the cerebral meninges, the cervical portion of the cord being usually the only part involved, but the entire dorsal sheath may be filled with pus from within the cranium. Traumatism of the occiput by vertebral dislocations, strains, and severe concussions may excite a leptomeningitis over a limited area, from which it may extend, or in which a virulent infection may find a suitable field for development. Surgical operations upon the spine, penetrating wounds, scalds, bedsores, and communication with adjoining suppurative foci may furnish the infection. Tuberculosis is a common cause, but the resulting meningitis is rather less acute. This is the case to a greater degree in syphilitic inflammation, which has a marked tendency also to remain localized.

**Morbid Anatomy.**—The disease is usually of wide extent, the infection traveling rapidly through the arachnoid spaces, and finding in the spinal fluid an excellent medium for its propagation and extension. Congestion of the pia, of the adjoining inner surfaces of the dura, and of the cord, marked by increased vascularization and an increase of spinal fluid, passes into inflammation, with dulness of the membranes, opacity, thickening, and an exudate, varying in color from opalescent to puriform, and of corresponding consistency. The microscope shows the diapedic elements of inflammation and often numerous bacteria, including at times the pneumococcus of Friedländer. Tubercles here correspond to their histological and bacterial characters on other serous surfaces. For a time the somewhat resistant pia covering of the cord and nerve-roots protects these structures, and especially is this true in the purulent form of the disease. Usually the periphery of the cord and the roots show the inflammatory invasion, with corresponding changes in the nerve-fibres, neuroglial framework, and vessels. In cases reaching a convalescent or chronic stage, adhesions form between the cord and the dura, obliterating the arachnoid space over more or less extensive areas, distorting the nerve-roots, and sometimes changing the outlines of the cord itself. If cord-softening has taken place as a result of the meningomyelitis, degenerations of its conduction tracts and localized destruction of its gray matter are found. Large quantities of spinal fluid usually mark these late cases, causing, with the irregular adhesions, a sacculated condition of the dura.

**Symptoms.**—The abrupt onset of the disease may be preceded by a day or two of malaise and slight anorexia, but sometimes no invasion period is present. A sharp chill is followed or attended by great pain in the back and darting pains around the body or down the limbs. In children vomiting is a common symptom, and roseolous may be present. Tenderness is at once developed over the spine. It is easily detected, when not a prominent complaint, by the use of a sponge dipped in hot water or by percussion. Spasm and rigidity of the muscles appear, causing stiffness of the neck and back, sometimes notable retraction of the head and vigorous opisthotonos. Fixation of the limbs upon the body is more or less marked, with a tendency to flexed attitudes. Retraction of the belly results from implication of the abdominal muscles. Sometimes difficulty of breathing is occasioned by involvement

of the chest muscles. Kernig's symptom is present. Dyspnea, Cheyne-Stokes respiration, and cardiac symptoms follow medullary implication. The cramps in the muscles are painful, and yet tenderness and hyperesthesia in the limbs prevent manipulations and passive movements. The rectum and bladder are the seat of similar spasms, which may cause constipation and retention of urine, with frequent annoying and ineffectual expulsive contractions of these viscera.

*Pulse and temperature* are feeble, sometimes being subnormal, sometimes increased, and more often divergent; for instance, a subnormal temperature with an accelerated pulse. The lack of uniformity in their range is especially valuable in diagnosis, even when the cerebrium is apparently not involved. A temperature of  $103^{\circ}$  F. is not uncommon. *Vaso-motor* paralysis is usually shown by the vivid, persistent, but slowly developed line which follows every stroke of the finger-nail upon the skin, and from the same cause the limbs may be congested and even slightly edematous. At first, for a day or two, *reflexes* are inclined to be increased and later may be wanting.

Cases which outlast the acute symptoms develop paralysis, anesthesia, atrophy, and contractures in proportion as the cord and nerve-roots are affected. Paraplegia may result, presenting the features of a cross-myelitis, with bladder- paresis, bedsores, increased reflexes, and spasticity. Symptoms vary with the location of the disease, but its tendency to involve the entire spinal apparatus is marked, and indications of its effect upon all spinal segments are to some degree present in a majority of instances. Some regions situated in the focus of the inflammatory action show early and emphatic involvement; those at a distance may be disturbed very little. Yet in some purulent cases, where the dural sheath is greatly distended through its entire length with the large accumulation, the pia protects the cord and nerve-roots from infection, so that pressure symptoms alone may be present.

*Course*.—Some cases terminate fatally within a day or two; others last a fortnight, and may then end fatally or recover. The nature and virulence of the infection are determining factors, as is the location of the disease. Extension upward or early involvement of the high levels of the cord tend to an early fatal issue. Complete recovery is rare and the conditions resulting from secondary myelitis are of long duration and usually last the life-time. The tubercular and syphilitic varieties, as already indicated, run their course less rapidly, and the latter is capable of material modification by treatment. Even rare tubercular cases may get well.

*Diagnosis*.—The diagnosis depends upon the rapid onset, the pain in the back, the radiating pains, the rigidity, the increase of pain on voluntary movement, the hyperesthesia, and the feeble temperature and pulse. From myelitis it is distinguished by the paralysis and lack of pain which characterizes the cord-lesion, but the frequent association of the two is to be kept in mind. Hemorrhage into the subdural space, owing to the irritation of the nerve-roots, presents very similar symptoms, but is extremely rapid in onset, usually following trauma or a strain, and develops meningitis in a short time thereafter. Hem-



orrhage into the spinal cord gives instantaneous symptoms, immediate paralysis, and may be practically devoid of pain. The rigid form of tetany may present a very close counterfeit, but its long duration, remissions, and amenability to spinal sedatives, the absence of spinal tenderness and shooting pains, the possible history of previous attacks, and the usual irritability from pressure upon the nerve-trunks and arteries should differentiate it. Tetanus may be mistaken for spinal meningitis. The early trismus, the excessive hyperesthesia, the fever of onset, the paroxysms of spasm, and the frequent history of traumatism point the way to diagnosis. Muscular rheumatism and strain present a very superficial resemblance. Spinal puncture and bacterial investigation are always in order.

**Prognosis.**—The outlook as to life is always serious and is grave in proportion to the suddenness of the onset, to the virulence of the infection, to the implication of the upper portion of the cord, and to the height of temperature. The estimate is also to be guided by the previous condition of health and the age of the patient, children and the aged quickly yielding to the disease. Traumatic and surgical infection is less serious than auto-infection. The possibility of the removal of sources of infection is of some importance as to ultimate results, provided the patient survives the acute stage. The late results, due, for the most part, to permanent changes in the cord, are usually beyond the hope of marked improvement.

**Treatment.**—Complete and absolute quiet is to be insisted upon. The patient should be kept upon the side or face, if it is possible to do so without increasing the cramps. The partial knee-elbow posture over a mound of firm pillows will often be found very comfortable, and at the same time will afford the best opportunity for local applications. A hot bath and pack at the onset with active catharsis have seemed to do good. Sedatives, especially spinal sedatives, are frequently required to control the spasms and anodynes to relieve the pains. Quincke's lumbar puncture has here the same indications as in the cerebrospinal form. Flexner's serum should be used in all cases presenting the meningococcus. Iodid of potassium and ergot are of little or no value. The ice-bag to the spine is one of the most serviceable measures, but is rarely tolerated long by the patient, and its intermittent application is useless. It should always be tried. As the active stage subsides, light cauterizations with the Paquelin apparatus, mild sinapisms applied for six or eight hours, and the hot-spray douche seems to assist the reparative efforts of nature. Cerebral symptoms usually mean the implication of the brain-coverings, the spinal features become of secondary importance, and the treatment is that of cerebrospinal meningitis. The paralysis, contractures, and other late results of the myelitis are to be managed in accordance with the rules of practice in that disease.

**Chronic Spinal Leptomeningitis.**—The chronic form of inflammation of the soft spinal membranes is usually the sequential stage of an acute attack, but may follow alcoholism, syphilis, or tuberculosis. Its existence as a primary affection is open to some doubt, but a very slowly

developed leptomeningitis may follow concussion, though it is impossible in such a case to exclude immediate slight histological injuries of which the later inflammation is a natural development. The formerly much used term "chronic meningitis," which was applied to every group of obscure subjective symptoms, however remotely referable to the spine, needs no mention. In *tubercle dorsalis* there is almost invariably a chronic meningitis especially affecting the posterior portion of the membranes and the posterior roots.

The symptoms are practically those of the acute form much reduced in intensity, and are dependent upon similar causes. Pain in the back predominates, and spasm is insignificant or absent. The radiating neuralgic pains are especially pronounced, and paresthesia are prominent. Their distribution depends upon the nerve-roots involved and the location of the inflammation, which is much more circumscribed than in the acute form. The late manifestations are those due to neuritis originating in the roots, and myelitic symptoms are comparatively infrequent.

The anatomy of the disease is very little known, as opportunity for post-mortem examination rarely occurs, but more or less extensive fibrous thickening, or adhesions between the pia and dura which constrict the nerve-roots, may be found, and may girdle the cord. Degeneration of the spinal nerves traversing the lesion is not rare, and this accounts for the herpetic and other cutaneous symptoms which are occasionally noted.

The prognosis will be guided mainly by the effect of treatment, but a complete recovery is very rare. Each case must be carefully estimated by itself.

The treatment in syphilitic cases consists in the heroic management of that disease, and iodide and mercury in small doses are also the most efficient drugs in non-luetic cases. General measures are of service, and persistent counterirritation over the spine, preferably by Paquet's caustery, is the most valuable local measure. Sometimes rest in bed and the ice-bag to the spine are of distinct value. Sedatives and analgesics are often required.

**Spinal Meningeal Hemorrhage.**—Spinal meningeal hemorrhage is either *extradural*, in the vertebral canal, or *subdural*, within the dural sheath. It is frequently associated with intracranial hemorrhage and with hemorrhage into the substance of the cord, but also occurs independently.

**Etiology.**—Meningeal hemorrhage occurs frequently at birth in protracted and difficult labors, and is then almost invariably associated with extensive hemorrhage within the skull. It has been considered under the cerebral palsies of childhood. Spontaneous hemorrhage is very rare, but occurs in adult life at all ages. Disease of the meningeal vessels is sometimes the immediate cause, but in the great majority of cases it is induced by traumatism. It may be caused by direct blows or falls upon the back, shock communicated through the lower limbs, vertebral fractures and dislocations, penetrating wounds and even by severe muscular spasm, as in tetanus, convulsions, and violent clonic. No doubt syphilis, arteriosclerosis, purpura, scurvy, and other hemorrhagic states favor it. The blood sometimes comes from a thrombo-arter-



year which has eroded the vertebra and ruptured into the spinal canal or dura, or from one situated on the vertebral or basilar arteries. Hemorrhage into the cerebral meninges may find its way below the foramen magnum, and in the same way a spinal hemorrhage may invade the cranium.

**Morbid Anatomy.**—In *extradural* cases the clot usually originates from the rich plexuses of veins that line the vertebral canal. It may be of considerable size and extend through the intervertebral foramina. The most common location is in the cervical region. The dura is stained and infiltrated, and the cord may exceptionally be compressed. Effusions of blood within the dura vary much in size. The blood usually comes from the pial vessels, and consequently, as a rule, involves the cord. Complete flooding of the dural sheath is almost always due to intracranial hemorrhage or rupture of an aneurysm. A small hemorrhage tends to remain localized and to surround the cord at the original point. It discolors and compresses the cord and after a few days produces inflammatory changes in the meninges. In the same way an annular myelitis may be induced.

**Symptoms.**—The symptoms are practically the same in both extradural and subdural hemorrhage. The onset is ordinarily abrupt and the early symptoms depend upon irritation of the meninges and nerve-roots. There is great pain in the back, which often radiates along the implicated nerves, girdling the body or running down the extremities. Tugging and formication are complained of, and paralytic symptoms below the level of the lesion, loss of power, and diminished sensation are induced. Bladder and bowel symptoms shortly appear. There is ordinarily some spinal rigidity, which may develop into opisthotonus, and convulsions are not infrequent. Symptoms are promptly developed. In crushing injuries, spinal fractures, and dislocations the cord is almost invariably injured, and hemorrhage, if present, adds very little to the symptoms. From the onset to the full development of the paralytic features from one or two to forty-eight hours, or even more, may be required. The symptoms then greatly resemble those of spinal meningitis, which usually is added after a few days, and its invasion is often marked by a distinct aggravation.

Cerebral symptoms are only present when the cranial contents are simultaneously affected. Death is likely to occur early when the symptoms have reached their height, or during the secondary meningitis. Hemorrhage in the cervical region is strangely and promptly fatal.

**Diagnosis.**—In cases of insidious onset without definite symptoms, the diagnosis at best can be but conjectural. When hemorrhage follows trauma, the distinguishing trait is a gradual development of the symptoms within a few hours. Injuries that affect the cord substance produce instantaneous loss of function, but a meningeal hemorrhage may be, and often is, associated with hemorrhage into the cord. Jacobi has also obtained blood by spinal puncture in two cases of injury of the spine.<sup>1</sup> From meningitis the chief distinction is the much more rapid

(<sup>1</sup>) *Amer. Jour. Med. Sciences*,<sup>2</sup> Oct., 1900.

development in hemorrhage and the history of a competent cause. The *limiting diagnosis* is taken up in subsequent chapters, to which the reader is now referred.

**Prognosis.**—The outlook is always most serious. As the paralytic features develop, there is a likelihood of death from interference with respiration by paralysis of the chest-muscles. The intense pain and suffering also serve to exhaust the patient. The first danger being passed, secondary inflammation is likely to terminate the case fatally. Hemorrhage in the cervical region is, of course, more ominous than when situated lower down. If the patient survives the first fortnight, improvement may be confidently expected, and this may practically be complete, though some disability remains, as a rule, and it may be of an extreme degree.

**Treatment.**—At first the most complete rest on the face or side with the spine elevated should be secured. An ice-bag to the back is a valuable measure if persistently and thoroughly used. Venesection to lower the blood-pressure has been used, but will not find many brave enough to employ it. Local wet cups with free flow of blood have also been employed, but are of doubtful value. Remedies that increase the coagulability of the blood may be exhibited, but ordinarily the flow of blood is of but a few moments' duration, and no time is given for their action. If the diagnosis is fairly certain, the spinal canal should be aseptically opened and the dorsal sheath also incised. The operation is now done with nothing to the gravity of the case, and has enabled the surgeon to remove clots with the best results. The secondary meningitis and the sequential palsies are to be treated on their own indications.

## CHAPTER II.

### INJURIES AND DISEASES OF SPINAL NERVES.

THE spinal nerves, unlike most of the cranial group, are both motor and sensory. In addition, they contain the vasomotor supply, and through them is exerted the trophic influence of the spinal centers over the peripheral apparatus. Their injury or disease is, therefore, marked by perversion or abolition of these functions, and gives rise to groups of symptoms anatomically coextensive with the distribution of the particular nerve or nerves involved. We should bear in mind that the fibers making up a nerve-trunk are cellular elements,—prolongations from cell-bodies, of which they form an integral and functionally essential part. When we injure a motor fiber in a nerve-trunk we injure a motor cell. In other words, we injure the lower motor neuron. We will first consider nerve injuries and diseases in a general way, and then the particular conditions which pertain to such states in special nerves.



**Division of Nerves.**—Spinal nerves are frequently divided by incised and bullet wounds, sometimes by crushing accidents, by simple and compound fractures, and rarely by dislocations. Causes acting more slowly may end in the destruction of a nerve, but a neuritis or degeneration is usually, if not always, first induced. After a nerve is divided the peripheral portion degenerates, and the process is called *secondary degeneration*.

The immediate symptoms are loss of motion, sensation, and muscular reflexes in the distribution of the nerve. Shortly afterward, within forty-eight hours, the muscles supplied by the injured nerve lose their tonicity and then progressively waste. Vasomotor paralysis appears and trophic disturbances in the cutaneous area of distribution are marked by a thin, shiny skin, with atrophic hairs, nails, and other epithelial structures. There is also a lowered vital resistance to infection, and healing processes are slow and faulty. Even the joints are sometimes affected, and bony growth in the young is retarded. Electrical stimulation through the nerve fails completely. The muscles lose their responsiveness to faradism, and the increased galvanic irritability which at first appears gradually subsides and is finally lost. The electrical changes constituting the reaction of degeneration are more fully described in Part I, page 46. In the extremities the opposed antagonist muscles then draw the joints into fixed, rigid positions. Muscular contractures develop and still further tend to deform the part.

The histological changes that take place in the distal portions of the divided nerve are as follows: The nuclei of the internodal nerve-cells swell, and their protoplasm becomes increased in quantity, but changed in character, as it no longer stains so actively as in health. The nuclei also become segmented, and with the increase in protoplasm encroach upon the myelin and displace it. The nerve-fiber then shows an irregular beading of the myelin, and at the points of greatest constriction the myelin finally separates transversely, and the axis-cylinder is divided at the same time and in the same way. This process takes place uniformly throughout the length of the divided nerve below the

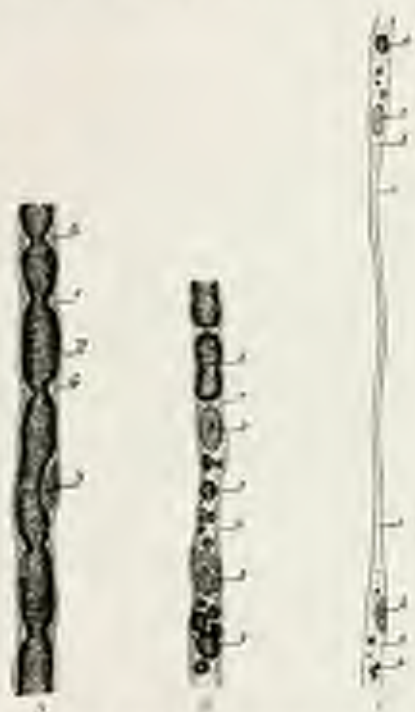


Fig. 132.—Nerve sections in peripheral portion of a divided nerve: A, two days after section; B, four days after section; C, eight days after section. 1, nucleus of internode; 2, myelin; 3, myelin sheath; 4, protoplasm (Kassirer).

lesion. The segmented myelin becomes more and more aqueous, escapes in part through its sheath, and is absorbed. The nuclei cease proliferation, and the nerve-fiber is left a mere connective-tissue filament, except at irregular intervals, where remaining globules of myelin may distend it.

The segmentation of the myelin in man reaches the point of complete division and consequent rupture of the axis-cylinder at about the end of the third day after nerve-division. At this time electrical conduction or excitability in the nerve is also lost and muscular tone is destroyed. The entire process of degeneration after nerve-division may be completed within three weeks.

Above the point of division the *central stump* degenerates for about a third of an inch only, but there is reason to believe that the cell-body is also disturbed (Marinero).

The muscles supplied by a divided motor nerve are deprived of the trophic influence of the spinal center as soon as division takes place, and the axis-cylinder below the lesion is equally deprived of the trophic support of the cell-body. The sarcolemma wastes. The cross striations become less well marked and are closer together; the muscle-fibers become narrowed, cloudy, granular, and sometimes fatty. There is proliferation of the connective-tissue elements of the muscles, which further strangles the muscle-cells, and eventually a condition of fibrosis or cirrhosis is developed. This has a natural tendency to contract and shorten, and explains the fibrous, tense, cord-like strictures and deformities found in such cases of long standing.

The electrical changes that occur in the muscle are also described in Part I, page 46. Faradic muscular contractions are lost within a few days, but at first, for about a week, the muscles react to galvanism even more freely than in health; then the galvanic excitability of the muscle is diminished and disappears.

When the ends of a divided nerve are brought together under proper conditions for healing, *regeneration* may occur in the peripheral portion even when degeneration is complete and of long standing. Beatty has noted one case of regeneration after fourteen years' division. In nerves freshly divided and at once united, the functions of the nerve are restored in a few days or weeks. After degeneration has been established regeneration is a slow process, requiring from two months to several years, depending upon the condition of the peripheral segment and the surgical features of the given case. According to Tizzone, Kennedy, Bethe, Ballance, and Stewart,<sup>3</sup> regeneration in a divided nerve is dependent upon proliferation of the neurolemma cells. At the end of three weeks spider-like neuroblasts are seen sending out beaded axis-cylinder processes in both directions from opposite poles. At the end of the fourth week these processes may overlap and anastomose. A new medullary sheath appears about the eleventh week. Neither axis-cylinder nor medullary sheath attains full maturity unless the distal segment is joined to the proximal nerve-stump. Non-myelinated nerves, as, for instance, cerebral conduction tracts, do not regenerate, lacking the necessary cellular elements required for that process. The muscles, in turn, regenerate, and slight voluntary motion returns before electrical

<sup>3</sup> Macmillan & Co., 1901.



responses can be obtained with the usual tests. These appear, as a rule, shortly after voluntary power is manifested, and while it is still very slight. Sensory conduction is usually restored before muscular action. The muscle reflexes are the last to reappear.

It need scarcely be added that the only *treatment* for a divided nerve is surgical suture. Whenever the distal extremity can be aseptically united to the central end, and in proportion as it is done promptly after the division, the prospects for a return of function are good. In cases marked by degeneration, electrical stimulation should be early and persistently employed, even if no motor response is obtained. In cases of long standing and in instances where it is impossible to effect nerve suture, the implantation of the distal segment into a neighboring nerve of similar function is frequently followed by a return of functional and volitional control.

**Neuritis.**—Peripheral nerves are subject to inflammatory action of every grade of severity, arising from an extraordinary number of causes. Many toxic conditions and cachexic cause widely distributed and usually bilateral symptoms, due to degenerative states in the peripheral nervous apparatus, producing a so-called *multiple neuritis*, the consideration of which is reserved for a separate chapter. We have here to deal with a nerve inflammation or degeneration arising from local causes. The particular cause, however, may be the typical feature of a systemic disease, as when a nerve is involved in a syphilitic tumor or a gouty, nodular thickening. Consequently the lesion in question is confined to one nerve or to several that are anatomically related.

**Etiology.**—Slight *compression* or *contusion* may cause temporary or persisting disturbance in the function of nerve-trunks,—a fact made familiar by knocking or pressing upon the ulnar at the elbow or by pressing on the sciatic in sitting. If the compression or contusion is of sufficient severity, the nerve may be an indefinite time in recovering, and long-continued pressure or extreme contusion may, like actual division, induce a complete degeneration. Dislocations, fractures, the formation of callus, violent muscular contractions, strains, cramped positions or continued pressure in sleep, coma, surgical anesthesia and extreme prostration, pressure from the continued use of surgical appliances, crutches, trusses, and constricting shoes or garments are among such causes. A nerve may be *wounded* or *infected* in surgical and accidental ways, and by the hypodermatic needle. It may be *wounded by extension* from a neighboring inflammation, as in arthritis, pleurisy, meningitis, bedsores, abscesses, or any other inflammatory focus. Exposure to *cold* is especially active, as in the facial nerve and others that are superficial or contained in rigid canals and passages. New growths, like cancer and gumma, and infiltrations in leukocythemia, cancer, leprosy, and syphilis may cause a neuritis.

There are also a number of *predisposing causes*, some of which are active in the etiology of multiple neuritis. Here may be enumerated gout, rheumatism, diabetes, exposure to cold, alcohol, lead, and cachexia generally. In such conditions an exciting cause acts more readily and repair takes place more tardily.

**Morbid Anatomy.**—The anatomical changes in a neuritis vary

according to the intensity of the process and the particular elements in the nerve-trunk that are most affected. When the nerve-fibers are mainly involved, we speak of a *perineuritic neuritis*. This form is rarely secondary to a local injury, but sometimes so arises. It is the common form in the multiple neuritis of systemic states. When the fibrous structure of the nerve-trunk is most affected, the neuritis is called *adventitious*. In the case of inflammatory extension from adjoining structures the sheath or surface of the nerve is usually first involved, and the condition is then distinguished as a *perineuritis*.

An acutely inflamed nerve looks reddened and presents increased vascularity. There is sometimes distinct edema, or a jelly-like infiltration of the sheath and adventitia may be seen. This is ordinarily confined to a limited extent of the nerve, or may be seen at numerous points. It is particularly likely to be present where the nerve is specifically placed over firm structures, as where a nerve passes over bony prominences or is tightly enveloped in fascial or bony tissues. From pressure or irritation thus arising, changes of corresponding degree take place in the nerve-fibers. When the neuritis or even the contusion is sufficiently severe to disrupt the myelin for a few hours, we have a descending degeneration below the lesion, which then presents the morbid appearances described in nerves after complete division. In the case of neuritis, however, it is common for some fibers to escape, and to appear practically normal in the cross-section of the degenerated nerve.

As a rule, the inflammatory and degenerative process is confined to the injured portion of the nerve and the parts beyond, but occasionally, and then usually in the case of infected wounds, the neuritis extends upward, and if it reaches the plexus of origin may there involve other nerve-trunks related to it. This ascending form is sometimes called *neuritis ascendens*. In cases of long standing the adventitia is increased and a fibrous thickening of the nerve may result, which persists even after restitution of function has taken place. In more chronic cases the degenerated nerve may be reduced to a mere fibrous filament.

Infiltration in syphilis and cancer is identical with these processes elsewhere, and may cause distinct tumors in or on the nerves. In leukocythemia and leprosy there is merely an infiltration, which in the latter may contain the characteristic bacilli. Perhaps the same may be true in neuritis arising from tubercular ulcerative lesions.

**Symptoms.**—The symptoms of neuritis vary greatly. They may be mainly those of *irritation* or those of *destruction* of the conducting functions of the nerve-fibers. Ordinarily both are present. Local conditions at the point of original nerve injury, such as swelling, contusion, or laceration, may be added. The onset is ordinarily abrupt, in the case of traumatic instantaneous. When the nerve is insidiously invaded the symptoms develop with corresponding slowness. The *sensory disturbances* are usually the most prominent at first. All varieties of paresthesia are complained of: Formication, numbness, tingling, burning, lancinating, deadness, coldness, etc. There is considerable pain in severe cases at the site of inflammation, and in the muscles and skin to which the nerve is distributed. The nerve-trunk and the supplied muscles are



usually tender on pressure. The nerve tenderness is particularly prominent where the trunk is rigidly held by fibrous tissues or passes over bones. The nerve may sometimes be palpably enlarged. Pressure upon it by muscular action may also elicit tenderness and increase the pain. The cutaneous area of its distribution presents hyperesthesia or diminished sensitiveness, or both. Often there is a feeling of painful sensitiveness when tactile perception is actually blunted. After degeneration starts in the nerve, anesthesia is present in the skin supplied by the nerve, though irritation at the site of original lesion may still lead to complaints of peripheral pain, constituting *anesthesia dolorosa*. The striking feature of all these sensory disturbances is their constancy and uniformity in a given case. Stabbing pains and momentary paresthesia are wanting. As the inflammation progresses the conductivity of the nerve decreases, hyperesthesia becomes anesthesia, and paresis passes into paralysis. The anatomical distribution of the sensory features is very important. Often it can be mapped out with the greatest precision, but at first usually the margin of the area of disturbed sensation is not so sharply defined and the entire limb may be painful and sensitive.

The *paralytic muscles* are those which are supplied by the given nerve. Opposing muscles may lack force from the loss of proper balance, or their use may be inhibited by pain. Delicate finger-motions are rendered clumsy by the sensory disturbance or the loss of power, or by both combined.

If the lesion be sufficiently severe to set up degenerative changes in the nerve, we find the *trophic losses* in the skin and muscles described under Division of Nerves, page 285. These may be, and often are, present in a modified degree when motor and sensory functions are not completely gone. In all cases of long standing they are sure to appear. With them go the various other symptoms already described: the loss of muscle reflexes, the electrical changes of irritation, degeneration or destruction, and the development of contractures. In complete division of nerves the *nutritional disturbances* is usually a simple atrophy, but in neuritis there is a wider range of dystrophic conditions. These are most marked in the hands and feet and most apparent in the dermal structures, though the muscular wasting is sufficiently apparent and often most striking. In some cases there is much obscuring edema, due to the lack of vascular and muscular tone. In these cases and in those in which redness and profuse perspiration are present, the dermal epithelium and the hairs and nails often take on an excessive growth. As a rule, the nails become roughened, scaly, and strongly curved. The cushions at the tips of the fingers waste and the nails may curve over toward the palmar surface, like claws. About the base of the nails the skin is often thin, glazed, and red, and the entire digit may be reduced to a tapering, stick-like appendage, closely covered with atrophic skin, which no longer shows the natural folds and wrinkles about the articulations. Abscesses do not heal readily. Herpetic eruptions are likely to appear, and deep ulceration may follow unless unusual care be exercised to prevent irritation and infection. In some cases the nails are cast off.

From inaction and the tendency to contracture in atrophied muscles and their supposed antagonists, the range of joint-motion in chronic cases is usually diminished. Trophic disturbances in the joints are also encountered, with the formation of adhesions, and false ankylosis. In rare cases joint-effusion and other neurotic *symplotheses* have been seen.

Should the neuritis ascend to the plexus and implicate other nerves, similar conditions are induced in their peripheral parts.

**Diagnosis.**—The diagnosis of a neuritis presents usually but little trouble. If the dysesthesia be confined to the anatomical limits of a given nerve or group of nerves, and the muscular disability is limited to the muscles innervated by the same nerve, the conclusion is obvious. Too much importance can not be given to the study of the cutaneous distribution of the sensory disturbance. If this area corresponds with that of any special nerve (see Figs. 15 and 16), suspicion of a neuritis or nerve injury should be at once aroused. If the corresponding reflexes are gone, or in slight and rare instances are even exaggerated, and if the degenerative or irritative electrical responses are present, the suspicion is confirmed. Sensitiveness of the paralytic muscles and of the supplying nerve-trunk are also important indications, and occasionally the thickened nerve can be felt. A wound or contusion over the nerve is of capital significance. The question often arises in traumatic cases as to whether the nerve has been completely divided. If the reaction of degeneration is present three or four weeks after the injury, the nerve is not totally divided. In the case of division no electrical responses are obtainable after a month. Many cases of neuralgia, joint tenderness, bursitis, myositis, and every variety of local pain or sensory disturbance are confounded with neuritis. The sensory features of neuritis are never intermittent, never fleeting, never diffuse. The anatomical distribution of sensory impairment of muscular implication and reflex alteration are very distinctive. Muscular wasting or electrical changes and altered reflexes demonstrate a neuritis.

**Prognosis.**—The course of a neuritis depends upon its cause and the amount of damage done the axis-cylinders of the nerve. The general tendency is to restoration of healthy function as soon as the cause is removed. A slight neuritis may pass away within two or three weeks, but if the lesion has resulted in degeneration of the nerve, months will be required for the regenerative process. The faradic muscle responses afford a good basis for estimating the probable duration of a case. If the muscles contract fairly to a moderate current two weeks after an acute attack, the disease will probably not last over a month or two. If they do not respond to a strong faradic shock, six to nine months will be required. In long-standing cases the persistence or reappearance of faradic excitability is a favorable sign for complete recovery. On an average, traumatic cases do better than those arising from extending inflammations, especially if they are of a septic character. Should there appear an upward extension of the neuritis the outlook is less favorable. The presence of any predisposing cause also diminishes the prospect of early recovery, and when the neuritis is due to a new growth the prognosis is unfavorable except in syphilitic cases.

Treatment is naturally first directed to any discoverable cause, and the



predisposing conditions must not be neglected. Gout, rheumatism, lead infection, nephritis, diabetes, tuberculosis, the use of alcohol, the presence of anemia, malaria, or any cachexia must be corrected as far as possible, and the general physical health brought to its best level. Wounds, septic inflammations, and new growths require surgical intervention. Pressure by crutches, clothing, and surgical appliances must be discontinued. Pain is ordinarily the most pressing complaint. It must be controlled as far as possible by securing complete rest, in severe cases by immobilizing the parts. Muscular action, ruble massage, and vigorous electrical stimulation produce pain, weak limbs, and should be avoided. On the other hand, gentle passive movements and muscle kneadings that do not cause discomfort, repeated daily or twice daily, are of distinct service in maintaining the nutrition of the parts that are temporarily deprived of their proper innervation. To this end the employment of electricity should be instituted, if possible from the first day. Ordinarily the galvanic current should be used, as it is least painful and most active. Only slight muscular contractions should be produced, and fatiguing the muscles must be avoided. If the faradic coil can be used without causing pain and the muscles respond to it, there is no objection to its use. The purpose to be kept in view is to maintain the responsiveness of the muscles to their weakened innervation, or, if that is cut off, to maintain them in the best possible nutritional and responsive state for the return of the slightest influence of the spinal cord that is able to first reach them through the blocked conduction of the injured nerve. These local measures also serve to overcome, so far as may be, the vaso-motor and trophic disturbances in the skin.

The use of analgesics calls for mature judgment. They should be postponed to the latest moment, as in cases demanding them a protracted attack is commonly in hand and the danger of installing a drug habit is very great. Counterirritation by sinapism, thermocautery, or small blisters over the nerve is useful in the early stages if it can be made reasonably near the point of inflammation. It should never be made in the cutaneous areas supplied by the affected nerve, as it is likely to produce uncontrollable ulceration.

In *chronic cases* massage, douching with hot and cold water, electricity, and counterirritation are of great value. Strychnin in large doses and hypodermatically sometimes is of use. The use of iodide or of mercury is of questionable utility unless indicated by constitutional conditions, though mercurial inunctions along the course of the nerve have seemed to assist in some refractory cases. Contractures and distorted joints should be treated orthopedically. Muscles that are wasted and weakened may be of some service if not placed at complete mechanical disadvantage by flexed joints and overacting, strong antagonists. Drooped foot, drooped wrist, flexed knee, and flexed hip should be obtained by early and persistent attention, by massage, and, if need be, by splints and fixation apparatus. Secondary contracture sometimes appears in the muscles of the extremities after regeneration of nerves injured by neuritis, just as it does in the face, but the balance is less delicate and dissection does not result so readily.

**Nerve-tumors.**—Spinal and cranial nerves may present new

growths of various sorts. In one group may be included those neoplasms which are common to all tissues, such as fibroma, myxoma, sarcoma, tubercle, syphiloma, carcinoma, etc. To another we should restrict those nerve-growths made up largely of nerve-fibers or nerve-cells, and these only may properly be called *neuronata*. In addition there are mixed and transition forms. In the first group the tumors are commonly sessile and usually single. In neuronata the tumors are commonly multiple and the condition is often hereditary and may be congenital or may follow traumatism.

Of neuronata a number of varieties are encountered which have received descriptive names. Those made up of ganglion-like cells are called *ganglion or cellular neuronata*. When the nerve-fiber elements are present the term *fibroid neuronata* is used, and this is further divided into the *myeloid* and *amyeloid* sorts, depending upon the presence or absence of the myelin constituent. The adjectives *dermatid*, *cutan*, *peripheral*, *multiple*, *trunked*, and *circoid* are merely descriptive of location or form.

Certain terminal neuronata which seem to be greatly enlarged Pacinian bodies constitute very painful tubercles under the skin, and to these the term *neuronata dolorosa* has been applied by Virchow.

The *multiple neuronata* constitute an important group. In some cases they number thousands, and vary in size from small pins to masses as large as a fist. They are located in three general ways: (1) They may appear along a single or along several nerve-trunks at somewhat regular intervals, like beads; (2) they may appear only on a single nerve and its branches, and (3) they may invade nearly every nerve in the body, including the sympathetic. In subcutaneous situations they appear like vascular, circoid dilations, but present a different consistency and anatomical position, are not pulsatile and are not modified by pressure on venous or arterial channels. They are most frequently acellular and devoid of myelin. While they may be sensitive and attended by neuralgic pains, they are usually unmarked by any sensory disturbance. As above indicated, such cases are sometimes of a hereditary nature.

*Traumatic neuronata* are rather common and their frequency after surgical operations has given rise to the term *amputation neuronata*. A nerve involved in scar-tissue by the healing process is likely to develop such a neuroma. In amputated limbs the nerve-stumps become clubbed, and upon examination there is found an increase in the nerve-fibers and axi-cylinders, which have a tendency to turn up and twist about in the small tumor, the size of which is relative to that of the nerve on which it develops. It appears to be but the thwarted natural effort of the central portion of a divided nerve to extend downward. Such traumatic neuronata are often exquisitely sensitive and may prevent the use of artificial limbs, besides causing much neuralgic suffering.

Neuronata in the limited sense of the term are benign growths, but occasionally after irritation or partial surgical removal sarcomatous conditions appear. From their number, and rarely from their position, as within the vertebral canal or cranium, or on the pneumogastric nerve, they may prove fatal. True neuronata are often associated with skin



tumors and cutaneous brownish circumscribed pigmentations. Preble and Hektoen<sup>1</sup> from the clinical side divide cases of multiple neuromata into four groups which may be variously combined in a given individual: (1) tumors of the skin, (2) tumors of the nerves, (3) pigmentation of the skin, (4) functional disturbances. This condition is frequently described under the title of Recklinghausen's disease, fibroma molluscum, or multiple neurofibromata of the skin.

**Etiology.**—In traumatic neuromata, and especially the amputation variety, the causation is readily understood. So is the action of irritation resulting in a fibrous proliferation which may strangle the nerve-fibrils, thrust them aside, or locate a fibroid thickening on or within the nerve-sheath. The hereditary features of multiple neuromata do not explain the initial liability to this affection. They also appear in myxedematous, cretinoid, and phthisical conditions, and, strangely, are almost confined to males.

**Symptoms.**—Neuromata manifest themselves by local signs and by motor and sensory symptoms in the distribution field of the invaded nerves. When situated on the nerve-trunk they may present much the same features as a chronic neuritis or local nerve-injury. Hyperesthesia, paresthesia, and motor and trophic loss are likely to appear with the electrical formula of degeneration. If the neuroma is open to palpation, it presents usually a rounded, dense, nodular, more or less sensitive swelling. Owing to its attachment to the nerve it has considerable lateral movement, but resistance is encountered in the opposite direction. Pressure sometimes provokes neuralgic pains or tingling in the sensory distribution of the nerve. Multiple neuromata, however, may be quite insensitive and present no sensory, motor, or trophic disturbances. They are only to be detected by the often visible chains of nodular enlargements along the course of the nerves. Traumatic neuromata usually can be readily palpated in the region of scars and give a feeling of shot-like bodies which are usually very sensitive. The rare, painful tubercles constituting the neuromata dolorosa variety are readily palpated and give rise to pain and tingling in the definite nerve-area allied to their anatomical location. In some instances neuromata have caused muscular spasm in their neighborhood, or even at some distance, and epileptiform convulsions have been cured by their removal.

**Diagnosis.**—Neuromata are readily diagnosed when a palpable, sensitive enlargement is found on a nerve-trunk with sensory and motor disturbances below. In cases of multiple neuromata the diagnosis is apparent. When single and deeply situated, their presence may only be inferred by the slowness of the onset of symptoms and the very chronic course of the disease. At the same time neuritis and pressure upon the nerve from adjoining new growths must, if possible, be excluded. In this case, also, it will be impossible to decide whether the growth in the nerve belongs to the first group or is a true neuroma. Multiple neuromata, traumatic neuromata, neuromata dolorosa, and neuromata occurring in myxedematous individuals are usually of the true and, hence, benign variety.

**Prognosis.**—Single neuromata are more likely to damage the nerve

<sup>1</sup> *Ann. Int. Med. Sciences*, Jan., 1902.

than the multiple variety. If the tumor is of some size and functional disturbance is absent, none is likely to develop, but if such symptoms occur, they are likely to increase. True neuromata are of slow development and present a long course. Malignant or specific growths in nerves present the same outlook as elsewhere.

**Treatment.**—The treatment of neuromata is practically surgical. They must be excised with as little damage to the nerve-trunk as possible. In the multiple forms surgical interference is hardly practicable except for isolated masses, or to relieve special nerves. If the tumor is confined to the nerve-sheath, it may be removed without much injury to the nerve-trunk, but if this is involved, the neuroma must be excised and the ends of the nerve sutured. This is now accomplished even when several inches of the nerve are removed, by interposing pieces of nerves removed from animals, or by entang. bundles, bone tubes, etc. There is, however, a decided liability to recurrence of neuromata after surgical interference, due to the preexisting tendency and the irritant conditions set up by operation and healing. Nerve-stretching is claimed to be more efficient in relieving the reflex spasms than excision. The use of anodynes for the relief of the neuralgic pains that sometimes make life burdensome should never be resorted to if the neuroma can be surgically dealt with. Pressure on the nerve above the tumor sometimes gives temporary relief from pain.



# CHAPTER III.

## LESSONS OF SPECIAL SPINAL NERVES.

ANY spinal nerve may be singly injured by trauma or disease, or several neighboring nerves may be involved at the same time by local conditions. Such lesions give rise mainly to disturbance of sensation, motion, and trophic control, showing themselves in varying degree in the cutaneous and muscular distribution of the injured nerves. The changed electrical conditions and the modified muscle reflexes that are also present equally depend upon the extent and nature of the lesion. These common lesions are division, neuritis, degeneration, and new growths, which have been considered in general terms in the preceding chapter. Irritant lesions produce morbidly exaggerated functions, such as spasms, hyperesthesia, pain, and rarely hypertrophy, while destructive lesions are marked by conditions of deficit, anesthesia, paralysis or paresis, dystrophy, and atrophy. These are often blended in the same case, as by the partial division of a nerve, or by a neuritis affecting mainly the motor or sensory portions of the nerve. For the sake of brevity, and to avoid repetition, these various nerve-lesions will be described systematically: (1) As to commonly acting causes; (2) as to the resulting motor disability and deformity, and (3) as to the sensory disturbance. A lesion of a disabling degree will be understood to be in operation unless otherwise indicated. Lesser injuries will, of course, present relatively diminished symptoms.

### THE CERVICAL AND BRACHIAL PLEXUSES.

The phrenic nerve, arising from the third, fourth, and fifth cervical nerves, is injured: (1) By disease of the cervical vertebrae or of the meninges or of the cord, affecting its spinal nuclei or roots. The condition is then usually bilateral, and other muscles than the diaphragm commonly suffer. (2) In the neck penetrating wounds may reach this nerve, or new growths injure it. (3) In the thorax tumors may compress it and inflammations extend to it.

The resulting motor loss consists of inactivity of the diaphragm on the affected side, which fails to descend on inspiration, and the corresponding portion of the abdominal wall does not advance equally with that of the sound side. This is especially noticeable on deep inspiratory efforts. When both plexuses are involved, the breathing is of a costal sort, and any exertion quickly causes respiratory distress. Difficulty in expectoration, sneezing, defecation, and other abdominal expulsive efforts is also present.

The sensory disturbance is obscure and often overlooked or misconstrued as intercostal neuralgia, muscular rheumatism, etc.

The posterior thoracic in its long course from the fifth, sixth, and seventh cervical nerves to its distribution in the serratus magnus is often subjected to mechanical pressure from heavy objects carried on the shoulder and by muscular compression in severe exertion or continued labor, particularly in overhead work. Mowing and tailoring also furnish cases. Penetrating wounds occasionally involve it, and falls or blows on the back may injure it. It also suffers in association with other nerves as a part of spinal atrophies. From the usual traumatic character of the disease, men in active middle life are most commonly affected, and on the right side, as a rule.

It occasions weakness in all the movements of the upper extremity that depend upon the fixation of the scapula and impairs thoracic in-



Fig. 345.—Dorsal plexus of the right serratus magnus.

spiratory expansion on the same side, but causes no absolute motor loss. The paralysis of the serratus causes a peculiar and characteristic deformity. Attempts to put the arm forward cause the posterior border of the scapula to widely wing out from the chest, so that a deep recess is formed behind the shoulder-blade. The upper portion of the bone moves outward and the lower angle toward the spine.

As the nerve is almost purely motor, the only sensory disturbance is neuralgic pain in the neck and shoulder in neuritic cases. The prognosis in injury to the posterior thoracic is comparatively less favorable than in other spinal nerves. A serratus paralysis is always of long duration and often permanent, even when there is every reason to believe that the condition has arisen from a simple pressure neuritis. In cases









PLATE I.



VERMICULAR AND BRANCHIAL NERVE (EYEWORM).





not open to nerve suture. Tubby<sup>1</sup> has suggested and successfully practiced the ingenious operation of substituting a portion of the pectoralis major muscle, which is dissected from its humeral implantation, split up, and inserted into the digitation of the serratus.

**The suprascapular nerve** arises from the fifth and sixth cervical nerves. It may suffer alone in shoulder dislocations. The supra- and infraspinati are paralyzed and the scapula becomes subcutaneous by their atrophy. The arm can not rotate outward at the shoulder, and there is a general lack of balance with weakness in the movements of the member. Carrying the hand from within outward, as in writing, is rendered especially uncertain and difficult. Anæsthesia over the outer portion of the scapula and the posterior portion of the deltoid is often present. Usually the suprascapular nerve and the circumflex are conjointly injured.

**The circumflex nerve**, arising from the fifth, sixth, and seventh cervical nerves, descends in the posterior cord of the brachial plexus, which it leaves to pass outward under the subscapular muscle, winds around the humerus, and is distributed to the *teres minor* and the *deltoid*. It also supplies the shoulder-joint. It furnishes sensation to the skin in a chevron-shaped area over the lower two-thirds of the deltoid. From its exposed position on the neck of the humerus and in the axilla it is often injured by shoulder dislocations, by arthritis, by crutch pressure, and by falls or blows on the shoulder. Injury of the circumflex causes loss of action on the part of the *deltoid*, and all attempts fail at lateral extension of the arm from the body. The loss of the *teres minor* action is insignificant.

Owing to the *deltoid* atrophy the acromion is uncovered and the shoulder rendered pointed and angular. The head of the humerus can readily be felt from the lateral aspect. In some instances it drops from its socket, leaving a deep furrow under the acromion. The nutrition of the joint also suffers and arthritis is likely to develop, limiting the range of joint motion. When the arm is passively moved, the scapula does not follow it unless joint disease is also present. An initial arthritis, by involving the articular branches, may spread to the circumflex and disable the *deltoid*.

Anæsthesia in the distribution field of the circumflex over the lower two-thirds of the *deltoid* is usually present.

**The musculospiral nerve** is the most frequently injured nerve in the arm, perhaps in the body. Arising from the posterior brachial cord, and



FIG. 100.—Atrophy of the *deltoid* and angularity of the shoulder in paralysis of the circumflex nerve.

<sup>1</sup> *Br. Med. Jour.*, Oct. 9, 1904.

arising originally from the fifth, sixth, seventh, and eighth cervical nerves or in some cases from the sixth, seventh, and eighth cervical and the first dorsal, it winds around the humerus in the musculospiral groove under the triceps, where it is subject to muscular compression and external violence or pressure. It supplies all the extensors of the elbow, wrist, and fingers, both the supinators, and through its radial branch the skin on the dorsal surface of the thumb and two radial fingers, and the posterior radial border of the hand. It also furnishes articular filaments to the wrist and carpal joints. By cutaneous branches given off above those to the triceps it supplies the skin in an area extending from the wrist in a narrow but widening strip up the dorsum of the forearm, and over the outer aspect of the arm as high as the insertion of the deltoid. These branches, however, are seldom involved in a musculospiral palsy. This nerve is injured in a variety of ways, and is especially involved in systemic states, such as lead poisoning. In those latter conditions it is interesting to note that the supinator longus, which is an active flexor of the elbow, does not participate. From its exposed position in the axilla, crutch pressure and dislocation of the humerus frequently affect it; lower down on the shaft of the humerus it is injured by fractures, nipped by callus, and subject to contusions from blows. Here it is frequently compressed injuriously by constricting cords about the arm, sometimes by violent action of the triceps, often by pressure during sleep with the arm under the body or resting on some hard object, as a chair-back, door-step, or bench. Such *sleep palsy* is sometimes presented after the pos-



Fig. 154.—Characteristic position of hand in musculospiral palsy.

ileged stupor of drunkenness, surgical anesthesia, or narcotism. Direct blows to the arm may also produce musculospiral palsy, and cold is often incriminated as a cause.

The *motor symptoms* of disease of the musculospiral nerve are extensive, interesting, and characteristic. The elbow and wrist can not be extended, and the long extensors of all the digits and the supinators of the hand are inactive after a lesion near the axilla. When the nerve is affected in the musculospiral groove, the usual location, the branches to the triceps escape and elbow extension is preserved. In lesions at or below the lower third of the humerus the branch to the



supinator longus escapes, as it does in systemic affections. The wrist can not be extended and wrist drop is produced. The fingers are semiflexed and can only be extended by the action of the interossei—which are supplied by the ulnar—after the first phalanges are passively extended on the metacarpals. The thumb lacks extension movements, and those of the fingers progressively diminish from the index to the little finger. Frequently on the back of the carpus there develops a *cystovial tumor* due to the overcarpal flexion, the inadequate support of the extensor tendons, and perhaps, in part, to the implication of the articular branches of the nerve. This constitutes a dense, painless elevation that exaggerates the wrist-drop deformity. Unhindered by the extensor paralysis the flexors are weakened so that the hand-grip is reduced in strength more than half. The muscular wasting shows most on the dorsal surface of the forearm. In these cases that involve the long supinator a very striking loss of contour is presented. The triceps may also show diminished proportions.



Fig. 115.—Dropped wrist from neurological palsy, showing cystovial tumor.

*Sensory disturbance*, in comparison with the extent of the paralysis, is very slight. Reference to the diagrams of cutaneous sensation (Figs. 15 and 16) will give an idea of the average space supplied by the radial branch of the musculospiral, but it varies widely in different individuals. The close relation with the median and ulnar also obscures the outlines of the field of disturbed sensation. One of the usual distributions of these nerves is indicated in figure 106. Often only prickling or slight numbness is felt in the tips of the thumb and index finger; in other cases the anesthesia is complete and sharply limited. In neuritic cases there is often complaint of constant pain in the wrist and carpal joints, which may be slightly swollen.

It is particularly in the treatment of dropped wrist that the great value of maintaining a proper position of the articulations may be emphasized. The tendency to carry the arm in a flexed attitude and the unopposed action of the flexors and pronators of the wrist give the carpal articulations a vicious position that is often difficult to overcome, and upon coalescence mechanically defeats the returning strength of the extensors. The use of a carefully padded and loosely applied anterior splint to maintain the wrist and fingers in line with the forearm hastens recovery in recent and in protracted cases.

**The median nerve**, originating in the sixth, seventh, eighth cervical and the first dorsal nerves, arises in front of the axillary artery by roots from the outer and inner cords of the brachial plexus. It follows the

brachial artery to the bend of the elbow, but gives off no branches above that joint. It supplies all the flexors on the front of the forearm except the flexor carpi ulnaris and the ulnar portion of the deep flexor of the fingers. It also supplies both pronators. In the hand it supplies the abductor, opponens, and short flexor of the thumb, and the first and second lumbrical muscles. These, like the interossei, are accessory to the flexors of the fingers for motions of flexion at the first joints, but are aids to the common extensor for extension of the second and third phalanges. The extensive distribution is subject to the differences pointed out above. In a general way we may say that the median supplies the



Fig. 106.—Distribution of sensory nerve on the back of the fingers.  
r, Musculospiral nerve; u, ulnar nerve; m, median nerve (Krym).

radial half of the hand on the palmar side and the tips of the index and middle fingers on the dorsal aspects. The median is rather rarely injured alone, but commonly suffers with the ulnar, or with the ulnar and musculospiral, from injury in or about the axilla. It may be divided by stab or bullet wounds in the arm, or fractures and wounds in the forearm, especially near the wrist, and by sprains or dislocations of the wrist. It has been torn across in compound Colles' fractures. Carrying heavy weights or holding objects for a long time in the bend of the elbow may set up a median neuritis by compression.

**Motor Symptoms.**—Disabling injury of this nerve destroys pronation, and the thumb can not be rolled into the hand nor opposed to the tip of the fingers. The fingers can not be flexed, excepting the ring and little fingers, which still act to the ulnar portion of the flexor profundus and the interossei. The wrist is flexed weakly to the ulnar side by the flexor carpi ulnaris. Through the action of the interossei the fingers can still be flexed at the metacarpophalangeal joints.

The resulting deformity consists in a flattening of the hand through loss of the thenar eminence, and the thumb lies in adduction parallel to the index in the same plane with the fingers, producing the "ape hand." This is also the usual deformity in progressive spinal muscular atrophy. The little finger retains all its lateral and other motions. The presence of the hypothenar eminence and the preservation of the ulnar side of the hand are distinctive. The swelling curve on the ulnar side of the forearm also disappears, and a concave outline may be perceived extending from the inner condyle to the wrist. From the action of the interossei there results a tendency to forward subluxation of the first phalange of the index and middle fingers at the metacarpophalangeal joints. Division of the nerve at the wrist only affects the thenar and first two lumbrical muscles and the cutaneous filaments.

The sensory disturbance is sometimes extremely slight, due to an unusually extensive distribution of the ulnar and radial nerves. It is most pronounced on the volar surface of the index, but may extend over the area shown in figure 107.

The ulnar nerve originates from the lowest cervical and the first



dorsal nerve, and supplies in the forearm the ulnar flexor of the wrist, the two inner divisions of the deep flexor of the fingers, and all the small muscles of the hand except those innervated by the median—namely, the dorsal and palmar interossei, the muscles of the hypothenar eminence, and the third and fourth lumbricales. It also supplies the adductor and one-half of the short flexor of the thumb. Its cutaneous branches supply the ulnar border of the hand, front and back, including all of the little finger, most of the ring finger, and a varying portion of the middle finger, largest on its dorsum.

The ulnar is (1) rarely injured above the elbow, excepting as a part of a more general injury to the brachial plexus. (2) At the elbow



Fig. 165.—Showing areas of sensory loss in injury of the median nerve (Flexor).

its exposed position behind the inner condyle and its superficial course through the forearm and at the wrist lay it liable to pressure neuritis and injury from wounds of all sorts. Even long-continued extreme flexion of the elbow-joint, as in sleep, in one predisposed, may suffice to induce a neuritis at this point. Occupations which necessitate continuous leaning on the elbow are also said to be active causes, but are certainly infrequent in this country. (3) Cuts at the wrist with various tools or from broken glass frequently divide it.

**Motor Symptoms.**—Ulnar paralysis constitutes a serious disability of the hand. Flexion at the metacarpophalangeal joints and extension of the second and third phalanges, which are dependent on the interossei and lumbricales, are lost. The wrist can not be actively flexed to the ulnar side, and the thumb is rotated toward the palm by the abductor and opponens and can not be adducted. The fingers lose all lateral motion.

The distortion and deformity that result are pronounced and characteristic. There is overextension at the metacarpophalangeal joints, which makes the head of the metacarpal bones prominent in the hollowed palm. The unopposed flexors "flaw" the second and third joints, and with the strongly-acting common extensor increase the deformity. This

is least marked in the index and middle fingers, which do not lose their lumbrical muscles. All the interosseous spaces are emptied, and the fifth metacarpal is left entirely subcutaneous. In place of the hypothenar prominence there is a deep hollow. The unaffected muscles of the ball of the thumb stand out prominently in contrast with the skeleton-like hand. The loss of sensation is confined to the indicated area of cutaneous distribution, but is only complete in the little finger. Figure 108 shows its distribution and degree.



Fig. 108.—showing sensory loss and sensory position in lesion of the ulnar nerve. (Shapley.)

#### COMBINED PALSIES OF THE NERVES OF THE ARM.

It is common for several nerves of the upper extremity to be injured at the same time, causing combined palsies presenting symptoms of corresponding extent. The circumflex, supraclavicular, and non-scutaneous may be affected simultaneously by disease of the fifth and sixth cervical nerves, from which they arise. This is usually caused by forcibly dragging the arm downward or upward, lacerating the anterior nerve-roots<sup>1</sup> and may occur at birth. In this lesion sensory disturbance is usually absent. A new growth or injury at a point between the scaleni muscles opposite the sixth cervical vertebra may affect both spinal nerves. At this point Erb found they could be simultaneously stimulated by electricity. The muscles involved are the deltoid, spinus, biceps, and brachialis anticus, and the sensory disturbance corresponds to the cutaneous distribution of the three nerves in question. Kennedy<sup>2</sup> reports some such birth accident instances greatly benefited by surgical operation. A cicatricial condition was found at Erb's point above the clavicle and outside the sternomastoid muscle; the nerves were resected and sutured with comparatively early restoration of motor function. This operation tends to become definitely established. For instance,

<sup>1</sup> Huot, Daval, et Guilan, "Rev. Neurolog.," Dec. 15, 1903.

<sup>2</sup> "Br. Med. Jour.," Feb. 7, 1903.



Taylor<sup>1</sup> fully reports on the anatomy of the condition with the technique of the operation and numerous case records. Operation is recom-



Fig. 108.—Shoulder palsy affecting the axillary, deltoid, biceps and brachialis muscles.

mended by him during the first or second year. Tully<sup>2</sup> has remedied the defective biceps by utilizing the outer portion of the triceps, which is freed from its implantation into the olecranon and mattressed into the bicipital tendon.



Fig. 109.—Brachial plexus injury. Area of anastomosis below axilla.

Injuries to the brachial plexus usually involve more than one nerve in the arm. Nerve injury arising from dislocations of the humerus and from strains on the arm usually implicate several nerves. Thus the median, ulnar, and musculospiral may be injured together, or a single nerve may suffer. In figure 110 is shown a case suffering from a

<sup>1</sup>"*Jour. A. M. A.*," Jan. 12, 1907.

<sup>2</sup>"*Br. Med. Jour.*," Oct. 17, 1902.

wrench of the arm received in alighting from a moving street-car. The ulnar, internal cutaneous, nerve of Wrisberg, and intercosto-humeral were simultaneously injured. The anesthetic area could be distinctly marked out. A similar combination of nerve-lesions occurs in birth-palsies from pulling on the arm or using a hook over the neck or in the axilla. Figure 111 shows the deformity in such a case: in a breech presentation attempts were made to draw the arm down from the side of the head and resulted in injury to the median, ulnar, and musculospiral.

Fractures of the humerus may involve the ulnar with the musculospiral. Fractures in the forearm may injure the radial and ulnar nerves, which, also, may be simultaneously implicated in wounds at the wrist.

In an ascending neuritis other associated nerves are likely to be affected if the inflammation reaches the plexus (neuritis migrans). This most frequently results from infected wounds. A neuritis traveling up the median will first invade the ulnar and musculocutaneous, and then the other nerves arising from the brachial plexus. The order in which the symptoms arise is the key to diagnosis.

Disease of the cervical vertebrae, as Pott's disease; of the meninges, as cervical polyneuritis; or disease of the cord, as syringomyelia, may involve the brachial nerves in varying groups.

**Neuritis of the brachial plexus** is a rather common condition that is usually mistaken for a neuralgia, angina pectoris, or a rheumatic trouble in the shoulder and arm. According to



FIG. 111.—Brachial palsy from birth-injury. See 11 lesions in axilla.

Gowers, who first carefully described it, brachial plexus neuritis is analogous to sciatic neuritis in the lower extremity.

**Causes.**—This disease of the brachial plexus is encountered usually after nubile life, and especially in rheumatic and gouty subjects. In the majority of cases there is a history of attacks of lumbago or sciatica and a strongly marked lithemic heredity. Women suffer more frequently than men.

**Symptoms.**—*Pain* is the first and constant complaint. It is often of rather sudden onset and is likely to come in paroxysms, especially at night, becoming more continuous, but still with exacerbations. Motion that stretches the plexus, such as elevating the arm, is sure to provoke it, and any considerable use of the member is inhibited by the pain to which it gives rise. The pain may begin first in the region of the plexus, in the



clavicular hollows, or under the scapula, but soon involves the entire arm, runs up into the neck, and frequently affects the side of the chest.

Upon manipulation one or all of the nerves in the arm will be found tender to the touch, and this tenderness can be traced through the axilla, under the clavicle, and into the scaleni muscles at the side of the neck. There is often tenderness over the scapula in the distribution of the suprascapular nerve. The skin over the arm is usually hyper-sensitive, and the constant dragging ache in the shoulder and arm is almost unendurable. The patient is constantly trying to find an easier position for the extremity, but without success. The tender muscles become flabby and wasted, and sometimes show slight degenerative reaction to electricity in certain groups. *Dermal, epithelial, and vasomotor* disturbances are not infrequent. The reflexes are diminished, rarely exaggerated.

**Diagnosis.**—The diagnosis rests on the continuous character of the pain, the tenderness of nerve-trunks, plexus, and roots, and the evidences of the neuritic process in the dermal changes, the vasomotor failure and the tendency to, if not the presence of, muscular atrophy below the elbow. Paresthetic features of a persistent nature in the fingers are almost invariably present. The presence of joint disease in old, gouty, and rheumatic patients may mislead as to the order of events, for the joint disturbance may be induced by the neuritis, or vice versa. The history must determine the point. Inflammation or injury of the subacromial bursa may easily be mistaken for a neuritis. It is marked by a distinctly painful point just below the tip of the acromion and by the pain caused when the elbow is abducted more than 10 or 15 degrees from the side of the body.

In this *larsitis* the pain is almost always referred to the arm above the elbow and there is neither paresthesia, objective sensory changes, atrophy, or trophic disturbance in the hand or fingers. The deep reflexes are never reduced or abolished.

A supernumerary cervical rib may cause symptoms of a brachial neuritis and actually induce such a neuritis. This condition, though congenital, usually causes trouble only in adult years. Carrying heavy burdens on the shoulder or in the hand produces pressure or tension



Fig. 111.—Scarring of the brachial plexus on left side with wasting of arm and shoulder.

of the nerve trunks as they pass over the rib, and commonly the subclavian artery is also compressed, adding arterial and circulatory features, such as interruption of the radial and brachial pulse in some instances upon downward traction or upward extension of the arm. The symptoms are commonly referred to the forearm, hand, and fingers, and trophic changes in the digits are generally observed in pronounced cases. The x-ray will usually show the rib. When unilateral, there is commonly an associated slight cervical scoliosis, and the presence of such ribs elongates the chest apices upward into the triangles of the neck. Their surgical removal is a difficult undertaking not devoid of danger, but yielding curative results in most cases.<sup>1</sup> Every case of protracted painful affection of the upper extremity with brachial plexus symptoms should lead to a search for cervical ribs.

**Prognosis.**—Brachial plexus neuritis is always a protracted malady requiring from three to eighteen months, or more. In old arthritic individuals it is especially intractable, but usually terminates in recovery. Relapses are likely to take place, and some slight disability in the way of pain or weakness is likely to persist permanently. The long maintenance of flexed positions, with the double tendency to joint disturbance furnished by the rheumatic element and the nerve-lesion, frequently result in a limitation of the range of motion at the shoulder and elbow. The wrist and smaller joints may also be affected and the use of the hand considerably impaired.

**Treatment.**—The treatment is that for neuritis, with especial attention to the gouty state. Repeated counterirritation on the side of the neck, in the clavicular fossæ, and on the inner aspect of the arm with blisters or the Paquelin cautery may be used from the first. The arm should be immobilized, except for the mildest daily passive movements, slight massage, and electricity when the tenderness and pain permit. Early in acute cases the very free use of salicylates not only gives relief to the pain, but may bring about a speedy cure.

### NERVES OF THE TRUNK.

The individual nerves supplied to the trunk by the thoracic and two upper lumbar pairs are rarely singly involved. A vertebral lesion or a wound may so affect them, but the motor loss is hardly appreciable unless several suffer simultaneously. The neuritic pain arising from Pott's disease and the girdling sensation in this malady and in *tubæ dorsalis* have great value from a diagnostic standpoint. There is also some truth in the lay apprehension that herpes zoster may be fatal if it completely girdles the trunk, as in such instances it almost always arises from some implication of the nerve-roots by spondylitis, malignant growth, or other serious local mischief. Though irritation of a nerve may cause a localized herpes eruption, in true zoster the lesion is an inflammatory disturbance of the posterior root ganglia (see page 336).

<sup>1</sup> Streissler, "Ergänze der Chirurgie," etc., Bd., p. 280, 1903.



## THE NERVES OF THE LOWER EXTREMITY.

The nerves of the lower extremity are much less frequently diseased than those of the arm, but are subject to special accidents by pressure on the lumbar and sacral plexuses within the body, as from parturition and abdominal and pelvic growths. Psoas abscess and inflammation of the pelvic viscera may also invade these plexuses.

The **anterior crural nerve** may be affected (1) within the abdomen, when the iliacus is affected and flexion of the thigh on the body is weakened, or (2) below Poupart's ligament. After a paralyzing lesion the extensors of the knee are inactive and wasted and the knee-jerk is abolished. Difficulty is experienced in advancing the foot, especially in mounting stairs. *Acroesthesia* extends from the groin to the inner side of the foot, involving the entire extremity except the buttock and a narrow strip down the back of the thigh, which below the knee spreads over the outer side of the calf and embraces the foot excepting on the inner margin (see diagrams, pp. 52 and 53). This nerve was observed to be affected seventeen times in 1000 patients in the lying-in hospital of Copenhagen, in all subsequent to parturition.<sup>1</sup> As shown by Byrnes,<sup>2</sup> this nerve is affected singly but rarely by neuritis or pressure injury. Gout, rheumatism, diabetes, infectious diseases, toxic and septic conditions, obstetric and surgical procedures, exposure, and occupational traumata are among the causes. Direct injury, inflammatory, extension from appendicitis, and pelvic disorders have also been reported.

The **external cutaneous branch** of the anterior crural supplying the outer side of the thigh seems especially subject to sensory disturbances. In gout, alcoholism, tobacco excesses, and sometimes as a lingering feature in multiple neuritis this area is sometimes exquisitely hyperæsthetic, so that the patient finds it inconvenient even to allow the weight of the trousers upon the skin, or is conscious of an acute tingling when he passes his hand over the thigh, or complains of various persistent paresthetic sensory disturbances. The condition is called *neuralgia paresthetica* and may be due to defective return circulation associated with varicose veins and hemorrhoids. It is usually a neuritis and the nerve has been found thickened at the point where it crosses the pelvic brim.

The **obturator nerve** has the same origin as the anterior crural and supplies mainly the adductors of the thigh and the hip- and knee-joints. Its paralytic interferes with movements that require approximation of the knees and impairs outward rotation of the thigh through weakness of the pectineus and external obturator. There is wasting on the inner aspect of the thigh. The sensory disturbance is confined to the hip- and knee-joints, which may also display trophic disturbance. The knee pain in *meralgia paresthetica* is a familiar complaint due to irritation of this nerve.

The **superior gluteal nerve** supplies the gluteus medius and minimus and the tensor vaginæ femoris. Its injury interferes with abduction, outward rotation, and circumduction of the thigh.

<sup>1</sup> "Centralbl. f. Gynæk.," No. 25, 1901.

<sup>2</sup> "Jour. Nervous and Mental Dis.," Dec., 1913.

The great sciatic nerve is diseased singly more frequently than any



Fig. 122.—Paralysis of sciatic (right); wasting of right thigh.

other nerve in the lower extremity. Its size and exposed position within the pelvis, at the sciatic notch, and behind the neck of the femur, and the exposed position of its branches, notably the external popliteal as it turns around the fibula, just below the knee, and the plantar branches in the sole of the foot, lay it especially liable to accidents. It supplies the flexors of the knee, which also assist in maintaining extension of the body on the hip, and all the muscles below the knee. In spite of its size and extensive muscular distribution, paralysis of the sciatic nerve is not so disabling as that of the anterior crural. The hip-joint can be fairly controlled by the muscles that escape, and the knee is kept in extension through the anterior crural. The whole extremity is then thrown forward on the knee and used as a peg-leg, as in the hemiplegic gait (see p. 214), but without rigidity.

The *monoplegia* that results from palsy of the great sciatic

occupies a narrow strip from the gluteal fold to the ham, where it spreads, embracing the outer side of the leg as far forward as the tibial crest, and all the ankle and foot except on the inner side. The wasting is marked on the back of the thigh and below the knee; the limb may be reduced to the last degree. Perforating ulcers on the sole of the foot, particularly at the ball of the great toe, are likely to develop.

The external division of the sciatic, the *external popliteal* or *peroneal* nerve, occupies an exposed position (1) in the ham and (2) below the knee on the outside of the neck of the fibula. It supplies the extensors of the foot, the long extensors of the toes and the peronei, and is analogous to the lower portion of the musculospiral in the arm. Its paralysis causes *foot-drop* and *toe-drop*. The muscular fullness on the anterior and outer aspect of the leg is lost; the uncovered crest of the tibia is extremely prominent. A tendency to equinovarus is produced that is often rendered a permanent deformity by contracture in the calf-muscles. The outer half of the front of the leg and the dorsum of the foot are anæsthetic.

The inner division of the sciatic, the *internal popliteal*, is analogous to the median and ulnar in the upper extremity. It supplies all the calf-muscles, the long flexors of the toes, and all the small muscles of





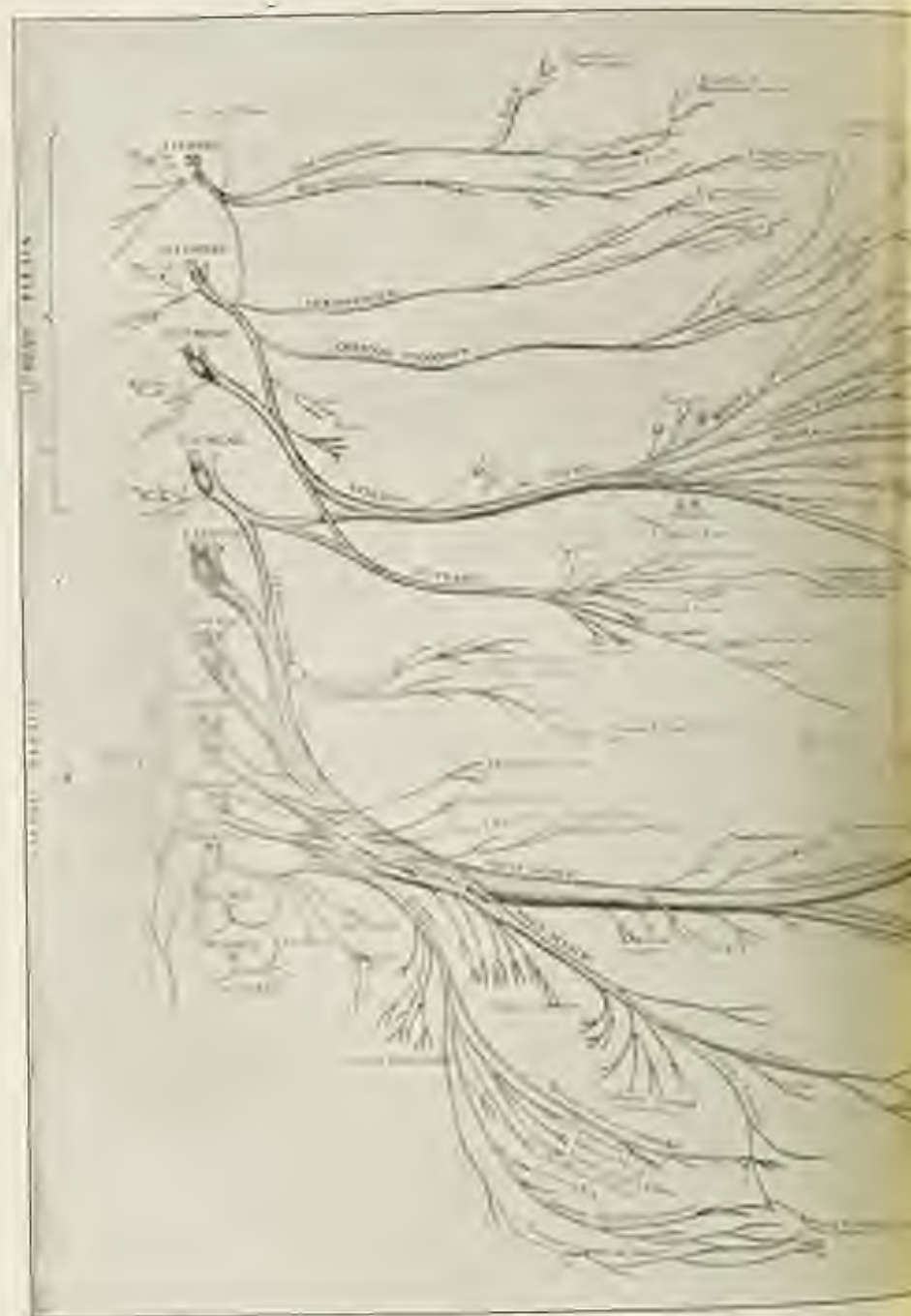


Diagram showing the Relations and Distribution of the Atlantic Ocean and the Gulf of Mexico



PLATE II.



of Lower and Sacral Nerves (Fischer)





the foot except the short extensor of the toes. It divides into the internal and external plantar nerves, which are sometimes singly diseased. When the *internal plantar*, which is analogous to the median, is alone divided, we have paralysis of the short flexor of the toes, the plantar muscles of the great toe except the adductor, and the two inner lumbricals. The big toe becomes overextended at the first joint and flexed at the second, producing the deformity called *hammer-toe*. There is anesthesia on the inner portion of the plantar surface, embracing also the plantar surface of three and a half toes.

When the *external plantar*, the analogue of the ulnar, is divided, the muscles of the little toe, the two outer lumbricals, the adductor hallucis, and all the interossei are paralyzed. All the toes assume the hammer-toe deformity, and the use of the foot is much weakened. Anesthesia is produced on the outer half of the sole and on the plantar surface of the little and one-half of the next toe. The dorsal surface of the foot and toes is supplied by the musculocutaneous and anterior tibial nerves from the peroneal.

When the internal popliteal is divided in the ham, in addition to the disability due to paralysis of both plantar nerves we have a loss of inward rotation of the flexed leg, due to paralysis of the popliteus muscle, and loss of power of extending the foot on the leg. By the overaction of the anterior leg-muscles talipes calcaneus is produced. The arch of the foot at the same time becomes exaggerated; the toes are extended at the first and flexed at the other joints, producing the analogue of the combined ulnar and median deformities in the hand.

*Morton's disease*, or *Morton's disease*, probably consists of a pressure neuritis or neuralgia of some, usually one, of the digital branches of the plantar nerves between the heads of the metatarsal bones. It commonly arises from the use of too narrow boots, and can be remedied in some cases by a shoe of proper width simply enlarging the arch of the foot. In the sole a depression is fashioned to relieve pressure on the particular painful point. Excision of the metatarsal head or neurotomy have both been employed successfully in severe cases.

**Sciatic Neuritis.**—Neuritic and neuralgic conditions have been so confused under the term sciatica that it is best avoided. Persistent pain confined to the region of the great sciatic nerve and its distribution, with tenderness in the nerve-trunk, is almost always due to neuritis. Sciatic neuralgia, on the other hand, is not a rare affection. Inflammation of the sciatic nerve, from its clinical importance, demands separate consideration.

**Causes.**—The causes of sciatic neuritis are those of neuritis elsewhere, and may be divided into those of a general and those of a local sort. In the first group we may name infant age, as sciatic neuritis is comparatively rare before twenty-one. Males are much more commonly affected than females, in the proportion of 8 to 1, according to Gibson.<sup>1</sup> Gouty, rheumatic, and syphilitic individuals are especially liable to develop it. Among *systemic poisons* that favor its occurrence may be mentioned lead, diabetes, syphilis, typhoid fever, malaria, and grip. *Pelvic disease* and *pelvic hyperemia* seem also active in causing sciatic neuritis, often acting also as local causes by transmitting pressure or inflammation

<sup>1</sup> "Lancet," London, 1880, No. 2622.

to the sacral plexus. It is most frequent in *wet* and *cold* seasons and among those whose occupations expose them to wet weather or extreme changes of temperature, such as stokers, puddlers, and laundresses. It may follow operations done in the lithotomy position.

Among the numerous *local causes cold* is usually considered most important. Sitting on damp ground or on cold stones, standing in water, or wearing wet clothing or shoes. *Overexposure* of the nerve in sitting or by vigorous action of the muscles and rarely *contusions* from blows below the sciatic notch may induce it. Bony thickening at the notch and syphilitic deposits at this site have occasioned a sciatic neuritis. In quite a proportion of cases there is an antecedent lumbago. The downward extension of the process in the muscles and fascia involves the sciatic nerve at the notch. All sorts of pelvic tumors and even constipation are capable of exercising such pressure on the sacral plexus that a sciatic neuritis follows. The nerve is sometimes injured in this position by the *obstetrical forceps* or through compression by the *grand uterus* and *fetal head*. *Spinal disease* and new growths within the spinal canal may give rise to neuritis by affecting the nerve-roots in the cauda equina or at the vertebral foramina. *Excessive fatigue* of the legs, as in the use of the sewing-machine, and the pressure of *varices* in the extremity or in the pelvis or about the nerve-trunk may occasion it.

William Bruce<sup>1</sup> takes the extreme ground that sciatica in practically every instance is due to *hip-joint disease*, an expression of some sort of arthritis, and more particularly of the upper and back portion of the capsule or acetabulum. The condition of the corresponding hip-joint should be examined in every case, but sciatica is no part of severe or destructive coxitis. The relation of sciatic disorder to *lumbosacral* and *iliac* distortion or disease has been given too much emphasis, but the condition of these joints in every case requires painstaking investigation.

**Morbid Anatomy.**—From rare autopsies and occasional operations the condition in the nerve is found to be a perineuritis, affecting also the adventitia. Secondly, the nerve-fibrils suffer. There is often an increased vascularity and redness of the nerve, and varicose dilatations in and about the nerve have been observed by some. The nerve is usually edematous and tumefied. In a word, we have to deal with an interstitial neuritis. Hunt<sup>2</sup> describes a perineurial jelly-like, quite structureless deposit in the nerve sheath unaccompanied by inflammatory changes and probably of gouty or rheumatic relationship. It is usually most marked at the sciatic notch and in the middle of the thigh, but may be more widely diffused. A similar condition is sometimes found in the branches, particularly the external popliteal.

**Symptoms.**—The primal symptoms of sciatic neuritis are *pain* and *tenderness*. While both have a tendency to extend throughout the sciatic territory, at first, and often during the entire case, they are confined to the cranial portion of the nerve and are most intense below the gluteal fold and in the upper half of the thigh directly in and over the nerve-trunk. The patient will often trace with his finger the seat of pain in a line that anatomically corresponds to the nerve itself. Below

<sup>1</sup>"Sciatica," Wood & Co., N. Y., 1913.

<sup>2</sup>"Am. Med.," April 11, 1905.



the knee the pain follows by preference the external popliteal nerve. The pain varies in degree, but is persistent in character, and even when slight is extremely wearing by its continuance. It is notably subject to exacerbations, which are quite likely to occur at night and seriously interfere with sleeping. Walking or any vigorous use of the limb is likely to increase the pain at once. Even after many hours of freedom from it, the pain may sometimes be promptly reestablished by taking a few steps. In bed the patient holds the limb by preference in a semi-flexed position at the hip and knee with the ankle extended. This obviates compression of the nerve by the muscles and avoids stretching the nerve by extension. If the nerve is stretched over the femoral neck, by extending the knee and flexing the thigh on the pelvis, pain is at once produced. This is known as Lesègue's sign.

These conditions give rise to a characteristic attitude and *gait*. The patient holds the knee semi-flexed and inclines the body to the opposite side, throwing into prominence the hip on the side of the sciatic neuritis. This tends to keep the weight on the sound limb, but its long continuance may develop a well-marked scoliosis with the lumbar spine convex to the neuritis and a compensatory dorsal curvature convex to the sound side. After recovery from the neuritis this *sciatic scoliosis* usually disappears. The *scoliosis* occurs in the opposite sense in cases marked by spasms in the affected leg and thigh, which are due to involvement of the sacral plexus or the lumbar cord, and manifest especially in the distribution of the anterior crural nerve. The hip on the side of the neuritis is then approximated to the lower ribs, the opposite one made prominent; the lumbar curve is concave, the dorsal curve convex, on the side of the disease. This *homologous sciatic scoliosis* is likely to be permanent inasmuch as the condition upon which it develops is a chronic one and contractures develop to fix the deformity. In walking there is an evident intention to use the diseased limb as little as possible in order to avoid tension and muscular compression of the inflamed nerve.

Pressure made with the fingers along the trunk of the nerve and its branches develops tenderness, which is greatest in certain regions. These correspond to the *tender points* of Valleix. The most constant are the following: The *gluteal point* over the sciatic notch, the *trochanteric point* above the trochanter major, a *trochanteric point* corresponding to the nerve-trunk on the posterior aspect of the thigh, a *popliteal point* in the ham at the division of the nerve, a *fibular point* where the external popliteal is superficial to the neck of the fibula, and a point on the dorsum of the foot. Frequently we find *lumbar points* just above the sacrum, an *iliac point* at the middle of the iliac crest, a *patellar point* over the knee-cap, points in the *calf*, points behind the *medals*, and *painful points* in the sole of the foot. Garat<sup>1</sup> and Rainier<sup>2</sup> insist that tenderness is always to be elicited by pressure on or at the side of the fifth lumbar spinous process. As a matter of fact in some cases tenderness is readily caused by pressure anywhere in the sciatic area, while in not a few instances the classical points of Valleix are not present. Barré states that deep tenderness of the adductor masses of the thigh elicited by

<sup>1</sup> Wiener med. Wochenschr., 1907, No. 23. <sup>2</sup> Neurolog. Centralblatt, 1909, No. 20.

pressure between thumb and fingers is always greater on the affected side.

The *paroxysms of pain* that commonly mark the course of the disease are often of a lancinating, boring, tearing, burning character that racks the patient and exceeds his power of description and endurance. They are usually provoked by use of the member, by an exposure to cold, or by some manipulation of the parts, but may come on spontaneously and even periodically, as in malaria. They often disturb sleep, probably from pressure or a continued cramped position.

*Cutaneous sensibility* is often modified. Hyperesthesia in the sciatic area is the rule at first, but in prolonged or severe cases gives place to anesthesia. Often the hypersensory disturbance is confined to small areas which correspond fairly well to the painful points previously indicated. Various paresthesias are encountered, as a feeling of cold, heat, prickling, fulness, formication, below the knee and in the foot and toes.



FIG. 34.—Contracture of leg, dorsum on right side; dorsum of foot and leg, same contracture with tendency to bend on hip, dorsum on left, foot forward.

*Motor disturbances* are less constant and present much variation. They usually only appear in the graver cases. Sometimes the member is shaken in a spasmodic manner during the painful paroxysms, and painful cramps in the calf-muscles may aggravate the patient's suffering. *Reflexes*: The knee-jerk is rarely exaggerated, usually it is diminished, and the Achilles tendon reflex is almost always greatly reduced or absent. Gilson<sup>7</sup> notes that the cremasteric reflex is usually exaggerated and the plantar may be increased, but the toe-sign of Babinski is never present. If the muscles waste, fibrillar twitching is frequently observed, and there is paresis or paralysis. In the rare spasmodic cases already mentioned, in which the plexus or cord is involved, the entire limb may be drawn up in a painful and protracted spasm upon the slightest cutaneous irritation, even by a breath of air or the contact of clothing.

Involvement of the vasomotor and trophic functions of the nerve in severe and protracted cases renders the limbs livid, cold to the touch, with a temperature reduced several degrees. Barré<sup>8</sup> states that even in moderate and recent cases the lower external area of the leg will be found cooler than the corresponding surface of the sound limb. The perspiration is usually reduced, but may be increased, and the dermal structures frequently suffer, as in

<sup>7</sup> *Edin. Med. Jour.*, No. 9, 1903.

<sup>8</sup> *Jour. Amer. Med. Assoc.*, p. 1097, April, 1905.



neuritis elsewhere. Scaliness, herpetiform eruptions, erythema, and acne sometimes appear, and perforating ulcer has been encountered. The muscles waste and show the reaction of degeneration to electricity. The wasting is most noticeable in the leg and buttock, but the short extensor of the toes is the most frequently involved, and this too early and in mild cases (Barré). The enlarged nerve may sometimes be felt. An increase of cutaneous fat may mask the muscular wasting.

A double sciatic neuritis is almost invariably due to spinal disease or pelvic disorder, to the suspicion of which it should always give rise.

**Diagnosis.**—The diagnosis of a sciatic neuritis is not ordinarily difficult. It is based on persistent pain and upon the presence of the anatomically located tenderness. Neuralgia gives rise to fleeting pain without tenderness or with relatively very slight tenderness, but early in neuritis this combination may also obtain. Disease of the hip-joint is sometimes mistaken for sciatic neuritis. Here joint tenderness and fixation and the location of the pain, which does not correspond to the sciatic, but to the obturator nerve, serve to differentiate the articular disease. Patrick<sup>1</sup> insists that with the heel of the affected side placed above the knee of the sound side, the patient in the dorsal decubitus, abduction and external rotation of the thigh will be limited in an affected hip-joint. Of course the presence of hip-joint disease does not exclude sciatic neuritis, but may even cause it. Hysteria sometimes mimics sciatic neuritis. The onset of the disorder under emotional stress, the absence of trophic disturbance, and the presence of contractures and other stigmata of the neurosis distinguish it. When the neuritis has resulted in wasted muscles, cutaneous eruptions, etc., it can hardly fail of recognition.

More difficult questions are those of the causation and location of the primal inflammation. In every instance a thorough investigation should be made of the pelvic organs and the spinal functions. If the neuritis is bilateral, the disease is almost surely above the sciatic notch. In talos sciatic pains are very frequent, but their lancinating character and appearance in storms, with other symptoms and signs of the disease, should lead to their proper classification. By the injection of cocaine into or close to the nerve at the sciatic notch the neuritic pain is inhibited, unless the trouble is higher up, and this measure, therefore, helps to locate it within or outside the pelvis. A history of syphilis, malaria, or climatic exposure sometimes tells the whole story.

**Prognosis.**—A pure sciatic neuritis is of good ultimate prognosis. If, however, it be due to pelvic or spinal disease, the outlook is modified for the worse by such conditions and in proportion to their gravity. As in any other neuritis, the probable duration must be estimated from the degree of severity of the neuritic process. The manageability of the patient has much to do with the prospects of a given case, as a great deal depends upon rest, and preferably rest in bed. Usually the disease lasts several months, and under conditions of use and irritation may extend over years or present numerous relapses. If nerve degeneration and muscular atrophy develop, from six to nine months will be required for recovery, though the pain may subside much sooner.

<sup>1</sup> J. Am. Assoc. Med. Assoc. December, 1917, p. 2176.

**Treatment.**—The general plan of treatment of a sciatic neuritis corresponds to that of any ordinary neuritis (p. 290), but certain local conditions require special attention. First of all and most important in the early stage is complete rest of the limb by rest in bed. Gouty, rheumatic, and other favoring conditions require appropriate attention, and, of course, pelvic and intestinal trouble must be properly treated. In acute cases 30 grains of aspirin or similar salicylate, with an equal amount of sodium bicarbonate, to protect the stomach, every hour for six days will often work wonders. During this intensive medication the patient must be kept absolutely in bed and under intelligent, watchful supervision. A little digitalis, strychnin, or caffeine may be added to support the heart. If good effects are secured, the salicylate in smaller doses at longer intervals must be kept up for several days.

Acupuncture over the course of the nerve serves the same purpose as any other form of counterirritation, and the same is true of injections of ether and other fluids. Gibson<sup>1</sup> advises puncturing the nerve-trunk itself with a good-sized needle in about five places, and reports, in 100 consecutive cases, 56 cured, 32 much improved, 10 improved, and 2 unimproved. The purpose of the nerve-puncture is to relieve the supposed edema within the sheath. The usefulness of the measure is, therefore, limited to early cases. For the same reason the needle should be used at the point of involvement, which is usually opposite the gluteal fold. When the nerve is thus pierced, a pain darts down its course. The needle is to be inserted at intervals of about an inch in the course of the nerve in the thigh, five times at a sitting, and repeated after two or three days. A somewhat similar treatment consists in puncturing the nerve near the sciatic notch with a hollow needle and injecting 100 to 150 cubic centimeters of a sterile normal salt solution. Cathelin's procedure of injecting the salt solution into the sacral canal is less painful, more easily accomplished, and gives equally good if not better results. This is the so-called epidural method.<sup>2</sup>

Negro<sup>3</sup> reports 113 rebellious cases in which vigorous pressure over the nerve at the most painful part resulted in recovery. He places the patient on his face, and, with all his force, presses both thumbs upon the nerve, rolling it from side to side for fifteen seconds. This is repeated after twenty minutes, and it is then much less painful than at first. The patient is relieved of pain for several hours and is enabled to walk. It is recommended that this manipulation be done about six times at two-hour intervals every second or third day. This procedure would seem better calculated to cause than to cure a neuritis, but is certainly valuable in chronic cases.

Vigorous massage in early cases has always seemed, in the writer's experience, to increase the pain and intensify the trouble, but in late cases is of great benefit, particularly in building up the wasted muscles. Electricity is also extensively used by some. When the inflammation is recent and the pain severe, a majority favor a broad, positive sponge-electrode over the nerve, with sufficient unbroken current to

<sup>1</sup> "Lancet," London, 1901, No. 3631.

<sup>2</sup> L. Struass, "Jour. Amer. Med. Assoc.," December 15, 1917, p. 2032.

<sup>3</sup> "Bulletin Méd. de Paris," Jan. 22, 1896.



red den the skin. In old cases the vigorous faradization of the thigh- and leg-muscles, though painful, sometimes seems to be of benefit, perhaps by the mechanical muscular pressure thus induced. High-frequency currents sometimes produce immediate and remarkably favorable results. So also do static sparks. Stretching the sciatic over the neck of the femur by forcibly flexing the hip-joint with the knee fully extended is also useful in late stages.

In most cases sedatives are required. Morphin is the only certain anodyne, but its disadvantages in the face of a protracted disease should postpone its use to the last moment. The coal-tar derivatives have some effect, and cocaine,  $\frac{1}{6}$  of a grain injected near the nerve, acts very well. The best results will probably follow complete rest and salicylates in the early stages; sciatic or epidural injection of normal salt solution in cases that do not promptly yield to rest and salicylates; and active massage, cutaneous stimulation, and free use of the limb in protracted cases.

Various springs and watering places have gained a reputation in the treatment of sciatic neuritis. All furnish hot baths and much rubbing or rude massage. The enforced idleness, abundant excretions due to drinking the water, frequent baths, and manipulations are the factors of their success. In early cases such a course is often clearly detrimental.

## CHAPTER IV. MULTIPLE NEURITIS.

UNDER the terms *multiple neuritis*, *multiple peripheral neuritis*, *polyneuritis*, *beri-beri*, *kaké*, etc., are embraced a number of conditions which have groups of symptoms mainly referable to disturbed functions of the peripheral nerves. The old-time distinction between central and peripheral organs and the clinical features of these cases directed attention mainly to the nerve-trunks and endings. In view of our present knowledge of the neuron unit, supported by numerous observations showing spinal nuclear involvement and even cerebral cortical disturbance in multiple neuritis, the term peripheral must be dropped. By multiple neuritis or polyneuritis is here meant a malady in which the anatomical lesions open to our present means of investigation are usually more pronounced in the nerves than in the central organs, and commonly consist of degeneration of the axis-cylinder process. Cases in which no histological change is found can not always be excluded, as many of the numerous poisons giving rise to the disease may, so far as we can detect, sometimes produce dynamic modifications alone. It is also allowable to suppose that the early effects of such poisons are perturbative of nerve-cell activity, to which the degeneration in the distant axis-cylinder process is secondary, and that if the poisoning be slight in degree, or the investigation be made before the secondary results are developed, no change whatever will be presented. Finer methods, however, are daily displacing post-mortem appearances which were formerly considered normal, and the dynamic cases are being steadily brought within the domain of the morphologically abnormal. It is likely that polyneuritis were better classed as a general disease of the nervous system. The preponderance of neuritic conditions has dictated its description in this connection.

**Etiology.**—Polyneuritis is the result of a systemic poisoning or of conditions which entail depraved nutrition, or of both. The toxic substances which are capable of producing a multiple neuritis are most commonly alcohol and lead; less frequently arsenic, mercury, silver, phosphorus, sulphid of carbon, acid of carbon, and ergot. In Manchester, England, an epidemic of multiple neuritis was traced to arsenic in the beer.<sup>1</sup> Certain metabolic substances may be developed that have the same pathogenic power, as in diabetes, nephritis, and intestinal disturbance.

The *infectious* furnish another group of causative agents. Most of the infectious fevers—typhoid, typhus, the exanthemata, erysipelas, purpural infections, acute rheumatism, influenza, and, above all, diphtheria—are occasionally followed by a multiple neuritis, which usually appears during convalescence. Even whooping-cough has been complicated by multiple neuritis.<sup>2</sup> In rare instances the neuritic disturbance appears

<sup>1</sup> J. S. Bury, "Br. Med. Jour.," Dec. 1, 1900.

<sup>2</sup> Collins, "Jour. Am. Med. Assoc.," Jan. 10, 1902.



early in these infectious diseases, and again rarely it may follow them after a considerable period of time, even after several months have intervened. The related toxins are assumed to be the determining agents.

Syphilis and tuberculosis have both been cited as causal of polyneuritis. Undoubtedly they both predispose to it by the systemic depression they occasion, and both are capable of producing a local neuritis by their specific proliferations. The anesthetic form of *leprosy* is frequently marked by a polyneuritis which is actually a peripheral disease and due to the invasion of the nerve-trunks by the bacillus *leproe*. *Malaria* can undoubtedly produce it. Eisenbar, in Hamburg, and Greene Hammond, in Connecticut, have reported small epidemics of the disease, which also in the form of *beriberi* is common in certain parts of Asia, South America, and on shipboard during long voyages.

*Obstacles* of all sorts, but especially those associated with cancer, are likely to develop polyneuritis; usually, however, combined with changes in the spinal cord. Old age, and especially old age marked by extreme arteriosclerosis and atheroma, may be the occasion of the disease by failure of nutrition both in the trophic spinal apparatus and in the peripheral nerve-trunks. Multiple neuritis occurs infrequently before adult age. In children it is usually due to infection, especially diphtheria, measles, and influenza. Much more rarely it is caused in childhood by lead or arsenic. Arsenic has caused polyneuritis in some cases of cholera where it has been too freely used. After adult age is reached all forms are comparatively common. Alcoholic polyneuritis is most frequent between thirty and forty. The senile and atheromatous forms appear only after sixty. Women furnish the greater proportion of alcoholic cases; men, of the rheumatic and toxic variety. *Occupation* plays a large part. Painters, plumbers, type-setters, rubber-workers, match-factory employees and employees in white-lead factories, and lead-miners are especially exposed. Various other pursuits requiring the use of lead, arsenic, and mercury, or the handling of spirituous liquors, the last by indulgence, furnish many cases. Very often more than one cause is operative, as when tuberculosis leads to constant alcoholic stimulation, or the onset of physical or mental depression in an alcoholic precipitates the attack of polyneuritis.

**Morbid Anatomy.**—*Lesions in the Nerves.*—In the very great majority of cases the condition found in the nerve-trunk is one of degeneration comparable to that caused by transection, with the difference that in polyneuritis the nerves show many axis-cylinders in a comparatively normal condition. Indeed, all grades of Wallerian degeneration may be encountered in a single nerve, the fibers being differentially affected by the toxic agent. It is also evident that the intensity of the toxic process and the duration of the disease will produce corresponding modifications in the nutrition of the axis-cylinder. It is possible that eventually we will be able to distinguish a variation in the degeneration produced by the various provocative agents, but at present they escape detection. The degeneration following lead-poisoning is apparently identical with that caused by toxins. The intensity of the neuritis generally decreases from the periphery toward the centers. Hypertrophy

of the endo- and perineurium is frequently observable. In addition to the axis-cylinder changes in some cases, usually those of long standing, there is an increase in the adventitia. A considerable thickening of the nerve-trunk results. This fibrous proliferation may even be excessive and at exposed points sometimes produces nodular enlargements. Syphilis, leprosy, and tubercular changes incident to these various diseases are also encountered. In the leprosy form the nerve may also contain the characteristic bacilli. Cenk,<sup>1</sup> Crocq,<sup>2</sup> and others, by inoculation experiments in lower animals, have produced the same changes in the nerves and in the spinal cord as are found in human subjects, but not in the brain.

*Lesions of the Spinal Cord.*—In a considerable and constantly growing number of cases the cellular parts and even the conduction tracts of the spinal cord have shown involvement. Oertel, Dejerine, and Pernice have described lesions of the anterior horns in diphtheric neuritis. Many, among them Finlay, Achard, Schaeffer, Larkin and Jelliffe,<sup>3</sup> and Clarke,<sup>4</sup> have noted the same thing in alcoholic neuritis. Others again, as Thöniich and Roennbach, recognize a simple atrophy of the cornal cells in lead-palsy. The cells of Clarke's column and the fibers of the posterior and cerebellar tracts have presented diffuse lesions of an inflammatory and degenerative nature. This observation has been confirmed by Herzog.<sup>5</sup> Henschen<sup>6</sup> records a case of typical diphtheric polyn neuritis followed by acute disseminate sclerosis. The spinal cellular changes consist either of a changed circled form with vacuolation, or of changes of an inflammatory character, sometimes with hemorrhages. The lesions of the spinal white matter are principally degenerative.

*Lesions of the Brain.*—Pernice and Scagliosi<sup>7</sup> in diphtheric cases found the principal changes in the brain, cerebellum, and cord, and referred them to circulatory disturbance followed by degenerative changes and hemorrhage. The cortical cells showed atrophic degeneration, which, as in the cord, especially affected their protoplasmic prolongations. The well-known effects of alcohol and lead on the cerebrum are also found. Delis,<sup>8</sup> experimenting on animals with poisonous doses of alcohol, found by appropriate stains that many of the cortical cells were changed in whole or in part. The presence of psychical symptoms in many cases of polyn neuritis abundantly proves that the affection is one that does not spare the highest nervous apparatus.

On the other hand, in many cases of polyn neuritis no changes have been detected. Thus Houché,<sup>9</sup> in a carefully examined case of widespread diphtheric palsy, found absolutely no change, even in the muscles which had been paralyzed. He, therefore, attributes the symptoms to toxins which operated without causing anatomical changes. The *spine nerve*, which is, in fact, a cerebral structure, is often affected in polyn neuritis. We have here to recall the whole list of toxic amblyopias. Alcohol again plays the most frequent part. The *medulla* and the

<sup>1</sup> *Brit. Med. J.*, Feb. 5, 1896.      <sup>2</sup> *Rev. Mes. des Mal. de l'Enfance*, Sept., 1893.

<sup>3</sup> *Med. Rec.*, July 8, 1899.      <sup>4</sup> *Br. Med. Jour.*, Sept. 12, 1903.

<sup>5</sup> *Deutsch. Zeitschr. für Nervenk. u. Psych.*, Bd. 37, 1909.

<sup>6</sup> *Fortschritte der Medizin*, 1896.      <sup>7</sup> *Brit. Med. J.*, Oct., 1893.

<sup>8</sup> *Centralblatt f. Nervenk. u. Psych.*, März, 1896.

<sup>9</sup> *Munch. med. Wochens.*, März 12, 1896.



*cranial-nerve roots* are subject to the same changes as the analogous portions of the spinal cord.

*Lesions in the Muscles.*—The muscles supplied by the affected nerves in polyneuritis undergo changes similar to those in simple neuritis or in nerve-division, but usually less in degree. The escape of numerous fibrils in the nerve-trunks is probably to be correlated with the persistence of numerous apparently normal fibers in the atrophied muscles. Another and important feature, however, is added, and that is the tendency to fibrous hyperplasia in the affected muscles, constituting in well-marked cases an *interstitial myositis*, which, in its turn, may act detrimentally upon the sarcolemmal elements. This muscular fibrosis also explains in part some of the tendinous contracture deformities of late cases. The presumption is that an element of irritation is present which acts through the apparently normal nerve-fibrils, though Babinski is disposed to attribute it to the pathogenic effect of the toxic agent directly on the muscle.

Changes in the trophic and secretory functions of the nerve are, like the motor and sensory, less pronounced than in a simple neuritis, but do appear, and are marked by disappearance of the fatty panniculus, by thinned and glossy skin, vascular disturbances, mottling, edema, lack of perspiration, and by modified growth of hair, nails, and epithelium.

**Symptoms.**—The symptoms arising from so wide-spread a disease are correspondingly varied. It is necessary to take them up systematically, but it may be said, once for all, that they are symmetrical, as a rule, affecting both sides of the body in a similar manner. This might be expected from a toxic cause of systemic distribution. It is, however, a fact that the symptoms usually commence first on one side and frequently are slightly more marked on one side than on the other throughout the disease. They may be alternately exaggerated on the two sides. Some toxic agents, as lead, affect more especially the upper extremities; others, particularly alcohol, principally involve the lower. Again, in some cases the motor symptoms predominate; in others, the sensory.

**Muscular and Motor Symptoms.**—*The Lower Extremities.*—The motor disturbance in polyneuritis is principally one of deficit, a paresis that in severe cases reaches complete paralysis. It is most marked at the distal extremities of the limbs, and in the great majority of cases affects first and most the extensor muscles. In the lower extremity the extensors of the toes, the peroneal muscles, and the dorsal flexors of the foot are the ones usually most implicated. In marked cases foot-drop is complete. The foot falls into line with the leg and the plantar surface turns inward, passively producing an equinovarus position when the patient is recumbent, or when the foot is raised from the floor (Figs. 115 and 116). In milder cases the patient can not raise the toes from the floor while standing on the heels, or, in other words, can not dorsally flex the foot beyond a right angle. The toes remain in a flexed, bunched position, and can not be extended or separated. Efforts against resistance at once disclose the weakness of these muscles. A peculiar and characteristic gait is developed. In order to clear the ground the patient is compelled to raise the foot by flexing the thigh. The leg is then thrown forward like a sail; the toe is pendulous; the outer border of the foot is depressed and brought to the ground toe first, or in a flat-footed

manner, or the outer border of the foot strikes the floor first. The other foot is then advanced in precisely the same manner. From this high knee-action Charcot denominated the gait "steppage." The dangling foot sometimes wears the boot at the toe and frequently scrapes up the dirt of the pavement. In very rare instances a contrary position of the foot and an opposite sort of gait has been produced by loss of power in the calf-muscles, the anterior group being spared. The



Fig. 11.—Drop-foot and Dropped foot in alcoholic multiple sclerosis.



Fig. 12.—Gait in multiple sclerosis, showing the high knee-action (and the dangling foot descending to its full and outer border).

patient in such cases in walking brings the heel first to the ground with the foot everted and dorsally flexed.

When the thigh-extensors are also affected and the muscles at the root of the limb are weakened, the use of the member becomes well-nigh impossible. A paresis of the extensors of the knee is often associated with that of the leg muscles in alcoholic polyneuritis. As above indicated, the loss of power may be trifling or absolute. While the muscle-groups enumerated are most affected, their opponents also lose power, but in



less degree. The loss of synergy alone would account for much of their weakness, but the neuritic process does not entirely spare them, and in severe cases of long standing they also waste.

*The Upper Extremities.*—The distal segments of the upper extremities, as in the lower limbs, are principally involved. The muscles of the forearm and hand are most affected. In polyneuritis the muscles under the control of the musculospiral are usually the first to suffer, the most involved, and the last to recover. *Drop-wrist*, the attitude characteristic of musculospiral palsy (see p. 299), is, therefore, a prominent deformity, and the disability of the hand conforms to this type, but the supinator longus often escapes. In some instances, to the drop-wrist is added the deformity characteristic of a median-nerve palsy, and the claw-hand or ape-hand is developed. The postcarpal tumor may also appear. In still rarer cases the deltoid, biceps, long supinator, spinati, and short supinator first suffer, but ordinarily they are involved, if affected at all, after the forearm and hand-muscles have been invaded. These functional groupings of the palsies of polyneuritis again furnish us with evidence of the essentially nuclear character of the lesion.

*The trunk-muscles* are sometimes also weakened, but only after the palsy of the extremities has become pronounced. Standing, walking, and even sitting may thereby be enfeebled or completely prevented.

*The muscles of the neck and face* in very severe and extensive cases may be affected, as may the muscles of the eyes, tongue, throat, larynx, diaphragm, of respiration, and of the heart itself. Involvement of the muscles of the throat, palate, and accommodative visual apparatus is a characteristic combination in diphtheric palsy. Branton<sup>1</sup> calls attention to a mask-like face, frequently seen in the alcoholic variety of multiple neuritis, affecting particularly the portion of the face between the eyes and lips.

*Electrical Changes.*—Owing, probably, to the variable amount of injury and irritation in the nerves, electrical tests in polyneuritis give all possible changes from the normal in different cases, and frequently at different periods in the same case. Not infrequently in the early days of the disease electrical excitability to faradic and galvanic currents is somewhat increased. This lasts but a short time, when diminished faradic response is presented, and may be attended in rare instances by voltaic diminution, a purely quantitative change. More frequently and customarily, as faradic excitability diminishes, the voltaic current augments in power and eventually presents some or all of the items of the reaction of degeneration (see p. 47). Complete abolition of electrical response to the galvanic current is, however, rare, even in the severest cases. Popoff<sup>2</sup> contends that diminution of electrical responses may be detected long before paresthesia is noticed. Erb has noticed that with the increased voltaic responses sometimes responses can be easily produced. A sharp blow across the muscles with the handle of a percussion hammer, especially near the tendinous portions, produces localized contraction of the muscle-fibers, causing a swelling that lasts for several minutes.

*Muscular atrophy* follows the muscular enfeeblement, which in its turn is usually preceded by sensory disturbances. The muscular tone

<sup>1</sup> *Br. Med. Jour.*, Dec. 1, 1900.

<sup>2</sup> *Neural. Centralbl.*, 1900.

sinks as the paralytic features develop and ankytropia is promptly added. In rare cases the loss of power is pronounced and yet the muscular masses retain their usual conformation and electrical responses, but gradually their swelling curves are replaced by straight lines or even by hollows, and the wasting in extreme cases apparently denudes the bones of everything but the dermal and fibrous structures. A polyneuritis may stop at any point or attain any degree of intensity. The muscular atrophy necessarily shows corresponding variations. As the paralytic features preponderate in the extremities and particularly in the distal portion of the limbs, the ankytropia is most marked in the same positions. We, therefore, find it ordinarily in the hands and feet, in the forearms and below the knee, but there is no voluntary muscle that may not be involved, and even the internal viscera are not immune. In some cases a thick layer of subcutaneous fat or an edematous condition may disguise the wasting of the muscles.

*Other Motor Disturbances.*—The lack of muscular balance in some cases and the diminution of muscular strength, whenever pronounced, may be attended by a certain amount of coarse *trembling*, which is likely to appear on voluntary effort or when the muscles are strongly exerted. This, taken with the disturbed sensation, especially in the fingers, leads to much clumsiness in the smaller coördinate movements of the hand. Most patients early find difficulty in picking up small objects, buttoning their clothing, etc. For similar reasons they are not so precise in the use of the lower extremities, and from the loss of sensation in the feet they swing and sometimes fall in attempting to stand with closed eyes. The gait may also appear *staccato*, but in the sitting or recumbent position these patients direct the movements of their lower extremities with as much exactness as their limited power will allow. The same is true for the movements of the upper limbs. The apparent ataxia must usually be attributed to muscular weakness. *Fibrillary trembling* in the atrophic muscles is frequently seen. *Cramps* sometimes occur upon effort being made after a period of repose, or spontaneously, or after the use of a comparatively mild faradic current. Patients very frequently complain of cramps, and of cramped positions. Hyperesthesia and other sensory disorders probably lead to such complaints.

*Contractures* are very prone to develop. Their disabling character can not be too strongly emphasized, the more so as the malposition to which they give rise can usually be prevented by proper, early management. They arise: (1) in part from the lack of balance in the muscles on the two sides of the limbs; (2) in part from the natural tendency of the flexors to impose upon the joints a position of *double flexion*; (3) in part from the action of gravity, and (4) in great part from the development of the muscular fibrosis and fibrotendinous retraction in the muscles incident to the disease. From these causes the foot-drop becomes a fixture. Attempts to dorsally flex the ankle-joint are stopped abruptly by the rigid, shortened heel-tendon, which alone impedes the joint's movements. A similar condition is often present at the knee, elbow, wrist, and even at the hip and shoulder. In exceptional cases tenotomies are eventually required to enable the patient to put the heels to the foot.

*The Reflexes.*—The cutaneous reflexes in the parts affected by a poly-



neuritis may be normal, but are usually diminished in activity and sometimes abolished. The *tendon reflexes* are almost always diminished or abolished in the limbs that are most affected, but, of course, only in the muscles that suffer. The Achilles jerk may be abolished and the knee-jerk present, or there may be some difference on the two sides of the body. In well-marked cases, however, the ankle, knee, wrist, and elbow reflexes are usually greatly diminished and often wanting. Occasionally in slight cases, and then usually only at first, the reflexes may be very active, or even exaggerated, but clonus is never encountered. The *organic reflexes* governing the intestine and bladder are very rarely impaired and practically never abolished. Sphincteric control is, therefore, maintained.

**Sensory Symptoms.**—The sensory disturbances of polyneuritis are usually the first to appear, the last to cease, and they often, even more than the paralytic features, distress and worry the patient. Popoff,<sup>2</sup> however, contends that reduced fadmic reactions are the earliest symptoms, and the author has fortold multiple neuritis in alcoholic cases by the diminished knee-jerks weeks before there was any complaint of paresthesia. Most cases begin by paresthesia in the extremities, which the patients describe by such words as tingling, numbness, dimbleness, pins and needles, coldness, heat, crawling, tickling, and similar expressions. They shortly notice that tactile impressions produce abnormal sensations, and it often requires careful questioning and examination to determine whether the condition is one of hyper-sensitiveness, blunted sensitiveness, or both combined. A light stroke with a feather may give rise to a distinct sensation of tingling, when the patient can not tell whether he has a small object, such as a pin, between the tips of his fingers and thumb, or may not be able to recognize such a familiar object by the sense of touch. As a rule, these sensory disturbances are persistent when once established, and only change gradually for the better or worse. In extent they present the greatest differences, depending upon the activity of the neuritis and its duration. Commencing usually in the finger-tips and in the toes and soles of the feet, they gradually extend up the limbs, enveloping them as if they were submerged in some loose material like cotton or shavings. Commonly the disturbance does not go above the elbow and knees, but may reach the roots of the limbs and invade the trunk. These areas of dysesthesia are not sharply bounded, but gradually merge into the normal fields. In a number of cases similar paresthesias are felt in the face, lips, and tongue at an early stage of the malady.

As the disease advances, these persistent subjective sensory symptoms become aggravated and frequently marked by crises of intense suffering, which are usually of the same character in a given case, but vary widely in different patients. One will complain of the most intense pain; another suffers with burning sensations and wants cold applied; a third feels that the extremities are cold to freezing and in a warm room bundles them with woollens. As a rule, when pain or sensory suffering is pronounced it is most marked in the lower extremities. It often deprives the patient of sleep and is an exhausting element in the case.

Sensibility to touch, pressure, painful impressions, and temperature may also undergo marked changes, which are infinitely varied. The

<sup>1</sup> *Nouvel. Centralbl.*, No. 11, 1903.

cutaneous sensations are disturbed in areas roughly corresponding to the parasthetic distribution and the motor loss. Early in these cases, when the irritative conditions in the nerve are probably at their maximum, there is a tendency to *hyperæsthesia*. The patients may complain that their stockings feel as though full of knots, that the bedding feels as if made of ropes, and in various phrases express the exaggerated modifications of touch. Light contact may be painful. Pressure on the nerve-trunks and on the muscular masses usually develop marked *tenderness*, and in severe cases may evoke a painful crisis. In many marked cases some dissociation of cutaneous sensations is observed. The pain and temperature sensations may be diminished more than tactile impressions, or touch may be abolished, the contact giving rise to pain or discomfort, which may not be properly located. In cases where the local conditions of wasting, *anæsthesia*, and electrical tests indicate marked nerve-degeneration, the patient may still complain of pains, burning, etc. In late cases *anæsthetic patches* are nearly always present, but it is rare for them to reach a complete degree. Some modifications of the muscular and joint sense are also encountered, so that the blindfolded patient may have uncertain knowledge of the position of the limbs. The transmission of all forms of cutaneous sensation may be slightly retarded, and generally all forms are uniformly decreased.

Various toxic substances differ in the sensory disturbances they produce. Some, with alcohol and grip at the head, produce marked painful disorders of sensation; while others, of which lead and diphtheria are the chief, mainly cause motor impairment,—another argument for the central location of the disturbing element.

**Ocular Symptoms.**—*Vision.*—Among the special senses, that of vision is the one principally, if not alone, affected. Many of the substances capable of producing a polyneuritis also engender a toxic amblyopia. These are especially alcohol, lead, arsenic, and the carbon gases. (For the special symptoms which pertain to them, reference should be made to p. 107, and to special works.) It must be borne in mind that even a pronounced optic papillitis, retracted fields, color *scotomata*, and considerably reduced vision may fail to attract a patient's attention and must be intelligently sought. In the toxic cases the condition is bilateral and symmetrical, and usually of insidious development.

*Motion.*—Squints and diplopia have been noted as frequently as once in ten times in diphtheritic neuritis (Rensak) and occur also, but less frequently, in the alcoholic form. They are due usually to a neuritis of the oculomotor nerves, but sometimes to nuclear involvement, when they are commonly attended with apoplectic features and are of sudden onset. The motor nerves of the eyeball may be affected equally or singly, producing corresponding deviations, *parésis*, etc. Commonly single muscles are paralyzed, a complete *ophtalmoplegia* being usually of nuclear origin.

*The Pupils.*—In rare cases of polyneuritis the pupils have been affected. Dilatation, contraction, and inequality have been noted by various observers. The pupillary reflexes, however, are generally unimpaired, and the Argyll-Robertson phenomenon is not seen. Brunsen contends that the pupillary response for accommodation is sometimes lost, that for light remaining active, the converse of the Argyll-Robertson phenomenon.



Accommodation is often disturbed in diplotheric neuritis, of which it is one of the most constant symptoms. Here, too, the pupils remain active. As a result of the paralysis of accommodation vision for near objects is reduced. This can be detected even in very young children by asking them to note a pin-hole in a calling card. For older patients printed characters are blurred and illegible.

**Trophic and General Conditions.**—In addition to the muscular atrophy, the trophic disturbance is manifest in the extremities, especially toward the digits. The skin is ill-nourished, cold, muddy in complexion, often turgid and reddened. It is often glossy about the nails and the fingers are tapered with overhanging, claw-like nails, which the patient sometimes refuses to trim owing to the tenderness and hyperæsthesia. Eczema is not infrequent and rarely an extra deposit of fat is encountered, but ordinarily the fat disappears early. Perspiration is reduced, or more rarely increased, causing a sodden crumbling of the epidermis. The nails and hairs may become coarse, stiff, and fragile. Herpetic eruptions, erythema, and ulcerations, so frequent in an ordinary neuritis, are very uncommon.

The general nutrition is usually greatly reduced and slight elevations of temperature are common. In the infectious forms fever may naturally be expected. The prolonged toxic or cachectic state, which usually antedates the onset of the polyneuritis, accounts for much of the physical depression, but some of it is apparently due, and its continuance is partly attributable, to the lowered functional activity that pervades the entire nervous system. In severe and prolonged cases difficulty in swallowing, an inactive stomach and intestine, a weakened heart and failing circulation, the continuance of pain, with perhaps insomnia and mental disturbance, produce a moribund depression that ends fatally or destroys resistive power against acute infections such as pneumonia. But usually the general state can be maintained at a satisfactory level.

**Mental Symptoms.**—Following Korsakoff, a number of writers have sought to delimit a psychosis peculiar to polyneuritis. It was pointed out by Cœda<sup>1</sup> that such mental disturbance occurred only when the patient was hereditarily neuropathic. Among others, Ferrari<sup>2</sup> has shown rather conclusively that this so-called polyneuritic psychosis is merely the mental state that marks chronic poisoning by alcohol, lead, and other toxic substances, and has something in common with the confusional insanity that sometimes follows infectious fevers. Clouston maintained that it was related solely to alcohol, but Bury<sup>3</sup> has noted it in the Manchester epidemic due to arsenic in very moderate beer drinkers. Even here, however, an alcoholic influence can not be denied. However, the association of mental disturbance with polyneuritis is extremely common and in slight degrees is easily overlooked. It is most often encountered in the alcoholic variety, then in lead cases and the infectious multiple neuritides. In some cases the mental condition precedes the appearance of neuritic phenomena, but more commonly follows the onset of the polyneuritis and develops progressively with it. It is first manifest

<sup>1</sup> "Revue Spéciale," vol. 23.

<sup>2</sup> "Allen and Searl," 2nd ed., 1893.

<sup>3</sup> *Ibid.*, 13.

in a change of temperament and character. The patient becomes irritable, capricious, and exacting, or may show apathy and indolence. This is attended or followed by loss of sleep and the nights are often marked by agitation and vague alarm, which subside with the day, to return at evening. Hallucinations develop as the madness advances and may lead, in the depressed and apprehensive mental state, to impulsive acts of a self-protective or defensive character. The patient may thus attack or abuse those about him or seek safety in flight. These marked mental symptoms may still subside toward morning, but finally in some cases the delirium persists night and day. When this cerebral condition occurs it usually appears comparatively early in the case, and corresponds, perhaps, to those irritative features that have been indicated in the peripheral nervous apparatus. Convulsions sometimes most clearly and emphatically demonstrate the cortical irritation.

As the paralytic features develop in the limbs, a partially demented condition obtains for the cerebrum. Often the period of *delirium* and excitement is not evolved, and the depressed mental condition is insidiously developed primarily. This is marked most prominently by a *forgetfulness* which pertains to the immediate past experiences and does not notably disturb the patient's recollection of events and conditions prior to the illness. Such a patient may talk rationally and logically on every possible subject, but is likely to forget what he has said or heard only a few moments previously, and repeats remarks and questions at short intervals or tells the same story over and over. Because of this amnesia he is frequently at fault as to the day of the week or month, or even the hour of the day, and may be unable to tell at noon what he had for breakfast. This is in most striking contrast with the patient's ability to remember the slightest details of his early life, with his apparent consciousness of his surroundings, and with his ability to logically associate facts presented to him.

In addition one often observes what at first would seem to be the play of a diseased imagination. These patients may assert, perhaps when already confined to bed for months, that they are just returned from a long journey, of which they give endless and minute details, name persons encountered, repeat conversations held and business transacted, when they were, in fact, unable to turn over in bed. Such fabrications are recounted in the most ordinary way, without the slightest excitement, and completely forgotten in a short time. If we analyze them, we find a mass of more or less definite incidents or purposes transferred from the patient's past life and brought down to the date at which they are recounted, the intervening time having disappeared. Patients will speak of having just seen relatives long dead, or give directions for the entertainment of persons who may be in distant parts of the world. If their attention is called to these discrepancies, they first try to maintain their statements, but readily become mentally confused and uncertain.

This amnesic mental condition may be developed in all grades of intensity, and may even proceed to complete dementia. The bodily strength and powers of assimilation usually are correspondingly depraved. The mental disorder passes away as the case mends and the



mind is usually perfectly clear before the peripheral apparatus is fully involved.

**Course and Special Forms.**—Polynuritic cases present many clinical varieties and some special forms. An ordinary case may be outlined as follows: The first complaint is of tingling or formication in the feet and legs, with occasional pains. After a few weeks the hands and forearms are similarly affected and some slight loss of muscular strength is recognized. The sensory and motor troubles increase. Pain becomes more prominent and the paralytic features invade the thighs, hips, and perhaps the trunk and arms. After about eight weeks the malady is full-blown. The patient now walks with the high action, the muscles of the extremities are notably wasted and give the electrical reaction of degeneration, foot-drop and wrist-drop are well marked, and the reflexes are diminished or abolished. The patient is uncertain on his feet, the legs and feet readily become edematous, red, and livid; with closed eyes he sways and perhaps falls. In bed, from the weakness in the arms and trunk, he can not gain the sitting posture without help. The muscles and nerve-trunks are sensitive to pressure; there is some anesthesia or at least much blunting and retardation of the cutaneous sensations, and much sensitiveness in certain cases. The skin is dry and thinned about the digits and base of the nails. At this time mental disturbance is likely to appear. After this state is reached the case remains almost stationary for several months; then the sensory disturbances diminish slowly, the pains decrease, the anesthesia disappears; the muscles regain their former proportions, strength, and reactions; the reflexes reappear, and full recovery is established, after about twelve months from the onset of the disease. Very often anesthetic areas become hyperesthetic and the pains increase as regeneration passes downward. In some instances the disease, instead of coming to a standstill, progresses to a fatality by involving the muscles of the trunk, of respiration, deglutition, and circulation. Death results from pneumogastric involvement. In other cases the development of fibrocartilagenous contractures disables the patient after convalescence until surgically relieved. Again, the progress of the disease may be much varied. It may commence insidiously and then advance with great rapidity, or, commencing suddenly, quickly reach its maximum and then slowly or quickly subside. When atrophy is well marked, restoration is always a tedious affair. As a rule, in the cases that recover full health is regained, but in a few cases localized atrophies and contractures remain permanently.

Other clinical varieties depend on the location of the prominent symptoms of the disease. The cranial nerves, the upper extremities, or the lower limbs may be most affected. Some cases show a preponderance of motor disability, others of sensory disturbance. The rare cases marked at first by increased reflexes, increased electrical responses, marked hyperesthesia, and perhaps a tendency to delirium, must not be overlooked.

In addition there is a small group of very acute cases in which the disease, beginning in the feet and legs, rapidly invades the thighs, the trunk, the upper extremities (commencing in the fingers and gradually passing up the limbs to the shoulder), and, finally involving the diaphragm and the bulbar nerves, reaches a fatal termination in from six

to twenty days. By some writers this form is considered identical with Landry's paralysis, but by others is distinguished from it by the disturbance of sensation and reflexes and the postmortem presence of changes in the nerve-trunks. Any sharp distinction is arbitrary and untenable, as all gradations between multiple neuritis and acute ascending myelitis are encountered.<sup>1</sup>

Certain toxic elements cause a polymyositis marked by peculiar and even characteristic symptom groups that merit brief attention.

The alcoholic form, besides being the most usual polyneuritis, presents with considerable uniformity the following features: The lower extremities are the most affected, and the painful sensory disturbances reach their highest range in this toxic state. They are proportionately much more pronounced than the paralytic conditions. The paresthesias are particularly intense and intolerable. The muscles and nerves are especially sensitive, and cutaneous hyperesthesia is exquisitely developed. The high-stepping gait is usually well marked, as the extensors of the leg are generally affected with the muscles below the knee. Atrophy is well marked and the tendency to fibrotendinous contracture is very strong. Involvement of the ocular muscles is comparatively frequent and bilateral exotropous anisotropia is common. Of all forms, alcoholic polyneuritis furnishes by far the greatest number of cases of mental disturbance. Recovery sometimes is comparatively rapid if alcohol can be withdrawn, but if the nerve degeneration is advanced a very tardy, lingering, and painful convalescence is the rule. Relapses are probable, and a single indulgence before the disease is definitely at an end may cause the most startling reappearance and exaggeration of all the former symptoms.

Lead-palsy in typical cases presents some or all of the following conditions: It usually comes on after prolonged exposure to intoxication through the patient's occupation, or by the use of drugs and cosmetics, or by drinking contaminated water. The source of the lead-poisoning is often extremely puzzling and requires the most painstaking search. The paralytic features are usually preceded by colics and constipation, and may develop at a long period of months after the patient has been removed from the source of intoxication. In other cases the administration of alkaline iodids to one impregnated with lead, but showing no marked symptoms of it, may precipitate the neuritis. The upper extremities are first and almost exclusively affected. In children, however, the lower extremities may suffer equally. The distribution of the palsy is practically symmetrical, but the right hand is most disabled in right-handed patients, the opposite hand in the left-handed. Sensory disturbance in the hands is practically absent, but if the legs are affected cutaneous sensibility is changed and pains are usually present below the knees. The muscular wasting is prominent, and in some cases seems to be the primal disorder, bringing these cases into apparent relation with the myopathies. Most cases show the wrist-drop feature, but arm and shoulder types may be encountered alone or may be gradually added to the first form, establishing an appearance of relationship with the spinal myopathies. The escape of the supinator longus, which is principally

<sup>1</sup> Meun, "Arch. de Méd. des Enfants," 1888.



a flexor of the elbow, while all the extensors supplied by the musculospiral are wasted, gives a distinctive contour to the forearm. The retrocarpal tumor is usually developed. The muscles of the larynx are frequently affected, as shown by aphonia. Ocular disturbances are frequent, and due to optic neuritis, from which sudden blindness may occur. When the trunk and bulbar muscles are involved, the outlook is better than in the alcoholic varieties under similar circumstances. The course is usually insidious at the beginning and protracted throughout. Anemia and basophilia are usually present, and a positive Wassermann reaction has been frequently found even in cases where syphilis could be reasonably excluded (Oliver).

Lead-palsy also has its associated mental disturbance. In rare cases this resembles the one so usual in alcoholic polyneuritis sketched at page 325, with marked visual hallucinations and amnesia. Lead, however, has its own cerebral disorders. These are an acute mania and a condition that presents all the somatic indications of general paresis, but is usually marked by mental sluggishness and is devoid of the expansiveness of true parietic dementia. In addition, lead may engender general cerebral affections by its action on the circulation, mainly by the arteriosclerosis and kidney disorders it induces. Delirium, convulsions, coma, hemiplegia, and their combinations are frequently encountered in plumbism.

**Diphtheric Paralysis.**—The term paralysis is selected purposely, as a neuritis can not always be demonstrated in these cases. Hochhaus found only the muscles affected, and Rolinski, in rabbits paralyzed by injections of the toxins derived from the Klebs-Löffler bacillus, found no changes whatever. In certain cases, however, a periaxial neuritis is clearly demonstrable. What the conditions are that determine a neuritis in one case and leave no trace in others can not now be stated. The result in both instances is undoubtedly due to the action of the toxins evolved in the life-history of the bacillus of diphtheria. Rosenfeld<sup>1</sup> calls particular attention to the increase of albumen in the spinal fluid and the lymphocytosis therein present, both of which improve with the general improvement. Diphtheric palsy may appear after all forms of the initial malady, whatever its apparent intensity or locality location. Warr, for instance, reports a typical case with the usual pharyngeal palsy, the diphtheric membrane being located on the vulva. It seems to be rather more common in adult cases, perhaps because the palatal, pharyngeal, and postmucogastic involvement in infants may lead to a fatal termination without being attributed to a nerve-lesion. Ordinarily, it appears during convalescence, in from one to three weeks after the diphtheria has subsided, but may develop within a day or two of the appearance of the pseudomembrane. The muscles of the soft palate and the pharynx are the first to suffer, as a rule, and the paralysis may extend to the lips, cheeks, and tongue. If it spreads further, the legs next suffer. The kneejerks are often abolished, even before the palate is disturbed. The upper extremities and the trunk are invaded only in exceptional cases. The muscles of the neck, on the contrary, are frequently weakened, so that the head may roll about helplessly on the shoulders. The affected

<sup>1</sup> *Deutsch. Zeitschr. f. Nervenk.,* Dec., 1908.

muscles rarely waste and contractures hardly ever develop. Sensory changes consist of insensibility in the affected parts, as in the pharynx, larynx, etc. Much pain and painful paresthesia are wanting. There is usually some ataxia in the extremities and Romberg's sign is seen. At the time the palatal difficulties appear there is usually some loss of visual acuity, especially for near objects, which is due to the characteristic paralysis of accommodation, but the pupils do not fail to act. Strabismus, double vision, and ptosis are not uncommon. In adults, sexual impotence is frequently developed. Mental symptoms almost never occur. Diphtheric palsy is of rapid development and usually terminates in recovery. If only the palate is affected, cure is reached in two or three weeks; in more generalized forms the disability is prolonged to three or four months, or even to a year in severe cases. If death occurs, it may be attributable to the cachexia and inanition from inability to swallow or may be caused by pneumogastric paralysis. Inspiration pneumonia is also likely to carry off the patient. In very rare cases of diphtheric palsy the paralytic features are fleeting, and change from place to place or disappear and return. Cases of diphtheric palsy are more frequent since the general use of antitoxin, probably because many cases are now saved that otherwise would have terminated fatally.

**Erythromelalgia** is a variety of multiple neuritis affecting principally the plantar nerves, and first described by Weir Mitchell as a vasomotor neurosis. Collier<sup>1</sup> reports ten cases of erythromelalgia in organic nervous disorders: six of insular sclerosis, three of tabes, and one of myelitis. It may also occur with decided arterial changes, but undoubtedly is a neuritis, as proven by Mitchell and Spiller.<sup>2</sup> It usually attacks men, and is worse at night and after walking. The feet become intensely red and show some swelling. The pain is of a burning sort and the attending perspiration may be profuse. Heat aggravates and cold relieves the distress. Elevation of the feet also affords relief. Walking may become so painful as to be impossible. Tropic disturbance in the skin in severe cases is encountered. The condition may get well spontaneously or be very persistent. Rest in bed, electricity, massage, and, in protracted cases, excision of the posterior tibial nerve have been advised.

**Beri-beri, or kakke**, is a polyneuritis that apparently depends upon a specific cause. Several investigators have found various micro-organisms which they have thought to be the pathogenic agents, but there is as yet a lack of uniformity and confirmation. There are those who insist that it is due to carbonic gas poisoning. Others have attributed it to a diet of fish or rice. Polished rice, or that denuded of the pericarp, is supposed to be particularly objectionable, the difference being attributed to the removal of the phosphorus compounds and the vitamins. It arises when people are huddled together in prisons, asylums, barracks, or on shipboard, and is particularly common in China, Japan, and India, and in South and Central America. Certain ships and prisons gain the reputation of being dangerous foci of infection. The growing tendency is distinctly toward ranking beri-beri etiologically as a deprivation disorder.

<sup>1</sup>"Lancet," Aug. 15, 1888.

<sup>2</sup>"Ann. Jour. Med. Sci.," Jan., 1888.



It is marked by paralytic and atrophic disorders, especially in the lower limbs, and particularly in the antero-external leg muscles, producing the characteristic gait. The phrenic and pneumogastric nerves suffer early, and respiratory and cardiac symptoms are prominent. The face and tongue are frequently affected. The sensory disorders are mainly anesthesia and severe, lightning-like pains. Edema of the lower extremities is often very great. It may also invade the trunk and flood its cavities. The knee-jerks are lost early. There is frequently right cardiac dilatation, rapid heart action, and murmurs over the base. Mental disturbance, similar to that in alcoholic cases, is frequently observed. All degrees of severity, from simple weakening of the lower extremities, with cardiac palpitation, to a pernicious acute form, like an acute ascending myelitis that runs to a fatal termination in a few days are presented. Intermediate varieties may be marked most by atrophy or by edema, and are very long in recovering, with death always likely from pneumogastric accidents. A large proportion of cases are said to have intestinal parasites. H. Wright<sup>1</sup> found changes in the cells of the posterior root ganglia and the anterior horns of the cord in eight consecutive cases.

**Leprous Neuritis.**—The invasion of the nerves by the leprous bacillus sets up a multiple neuritis or more properly a polyneuritis. There is a marked tendency to fibroid proliferation in the nerve-trunks, which often become nodular, and eventually the bacilli disappear. The spinal cord is exceptionally invaded by the bacillus (Souza, Martins), and presents central cavities, especially in the posterior horns and gray commissure. The prominent sensory disturbance is anesthesia, which occurs in discrete plaques, favoring in location the hands, feet, forearms, legs, and face. By spreading, an entire member or a large portion of the body may become anesthetic, and the deep parts, muscles, bones, etc., are also involved. Muscular atrophies are comparatively slight, and most affect the small muscles of the hands, feet, and face. Atrophic conditions in the hands and feet lead to mutilations of the extremities. Fingers, toes, and even hands and feet necrose and are cast off. The evolution of the neuritis is extremely slow, and may extend over a score of years.

In some cases a dissociation of cutaneous sensation is encountered similar to that of syringomyelia, especially of the Morvan type, and probably due to the cord lesions previously noted.

Recurrent multiple neuritis as a variety has been described by Sherwood, Ross, Dreschfeld, Targlowa, Klumpke, and Osler. Thomas<sup>2</sup> also reports a case. It appears that certain individuals are susceptible to repeated attacks from various poisons, but particularly from lead and alcohol. It is not requisite that the patient be exposed to the intoxicant to induce a recurrence of the neuritis, and it is undetermined whether there is an original susceptibility or the first attack leaves a predisposition to its return.

**Diagnosis.**—The diagnosis of a case of well-marked multiple neu-

<sup>1</sup> Brit. Med. Jour., June 29, 1902.

<sup>2</sup> Phila. Med. Jour., May 13, 1898.

ritis, based upon a fairly full history and a detailed examination, can hardly be missed; but in the early stages, when a diagnosis is of most importance, it is frequently extremely difficult. The major items upon which it then rests are the motor and sensory symptoms, their symmetrical distribution, their predominance in the ends of the extremities, the more marked affection of the extensors, the modification of the reflexes, the tenderness of nerve-trunks and muscles, and the history or presence of some toxic agent capable of producing the neuritis. A multiple neuritis may be confounded with several diseases of the spinal cord, especially poliomyelitis anterior and locomotor ataxia. The following differential tables may help to distinguish them:

## POLIOMYELITIS.

Most frequent in children.  
Onset abrupt.  
Endeavors to rise limbs.  
Not usually symmetrical.  
Immediate tendency to improvement.  
Sensory symptoms slight or absent.  
No mental symptoms.  
Usually leaves some deformity.

## LOCOMOTOR ATAXIA.

Girdle pains and lightning pains early.  
Nerve-trunks often insensitive.  
Muscular sense disturbed early.  
Anisotrophic and reaction of degeneration absent.  
Vivacity of gait due to hyperreflexia and irrespective of muscular strength.  
Strikes heels first and does not follow straight line.  
Circulation and trophic condition of limbs normal.  
Perforating ulcers, joint lesions, and osteomyelitis are common.  
Argyll Robertson phenomenon usual.  
Optic atrophy common.  
Vocal troubles frequent and early.  
Gastric and intestinal crises.  
Focal incontinence common.  
Sometimes followed by pure dementia.  
Of slow evolution, requiring years.  
Inevitable.  
Syphilis usually in the history.  
Spinal fluid characteristic.

## MULTIPLE NEURITIS.

In adults.  
Insidious.  
Begins in ends of limbs.  
Symmetrical.  
Gradual extension.  
Sensory disturbances early and persistent.  
Mental symptoms common.  
Recovers completely.

## MULTIPLE NEURITIS.

No girdle pains; lightning pains infrequent.  
Usually accompanied and often thickened.  
Only slightly disturbed or intact.  
Develop early.  
Due to poison and proportional to loss of power.  
Strikes toes and outer border of foot first and walks in straight line.  
Muscular atrophy, oedema, lividity, and epithelial changes.  
Rare or unknown.  
Never present.  
Rare, but toxic sensitivities frequent.  
Very exceptional and late.  
Dyspepsia from toxic causes, constipation from lead, etc.  
Only in acute perniciosa cases and in stuporous states.  
Often accompanied by mental disturbance.  
Of insidious, progressive development, requiring months.  
Reverses if patient survives.  
Antecedent intoxications, infections, and cachexias.  
Spinal fluid not distinctive.

*Myelitis* is distinguished from polyneuritis by the girdle pains, the paræsthesia, the anæsthesias, the retention of electrical responses in many of the paralyzed muscles, and the presence of the deep reflexes, which



usually are extremely exaggerated. In this disease bedsores are common; in polyneuritis well-nigh unknown. The sphincters are usually beyond ordinary control in myelitis and practically unaffected in neuritis.

*Hysteria* is sometimes confounded with polyneuritis, and, indeed, both may be present in the same patient, but should none the less be separately recognized. The pain in hysteria lacks sincerity of facial expression and the deep muscular masses are practically never sensitive. Hysterical anesthesia is segmental, and rarely involves all four extremities symmetrically. The reflexes and electrical reactions are present in hysteria—there is no wasting, no cutaneous dystrophy. The evolution of hysterical trouble, its emotional concomitants, and its stigmata are sufficiently significant.

The *pernicious anemias* often present reduced reflexes, paresthesias in the extremities, uncertain gait, mental vagueness, and general muscular weakness. A careful blood examination shows characteristic cellular changes (see page 468).

The *diagnosis* of the toxic cause and its origin is usually made in securing the history or in making the examination of the patient, but many times presents great and even insuperable difficulties. The alcoholic, lead, and diphtheric types of polyneuritis are themselves significant of their origin. Alcoholic addiction is frequently denied, especially by women. Patients may even be unaware that they are taking large quantities of alcohol in the form of various nostrums and patent medicines. The absence of the lead line on the gums should not mislead, as it only occurs where there is some local disturbance about the necks of the teeth. A dose of iodid, followed by an analysis of the urine, may show plenty of lead. Hair-dyes, face-powders, and styptic applications or injections may contain lead, and the polyneuritis may appear several months after their discontinuance. Arsenic and lead in fabrics, paints, wall-paper, toys, and medicinal prescriptions are to be borne in mind. In diphtheria, if the patient has been exposed, it is not necessary to have a pseudomembrane or even a history of a sore throat in order to attribute the palsy to the proper source. Leprosy and beriberi are distinguished by their infectious and clinical peculiarities. There remains the great class of infections arising from intestinal fermentation and the infectious diseases, which must be deciphered from the history and examination of the patient.

**Prognosis.**—It may be laid down as a general rule that if the cause of a polyneuritis can be removed, the patient has a very good chance of complete recovery, and that recovery is the most common termination of the disease. The prognosis is to be modified in accordance with the nature of the pathogenic agent, the mode of evolution of the disease, its intensity, extent, and localization, and the general physical condition of the patient. *Diphtheric* polyneuritis, unless the patient succumbs in the early days of the attack, is the most benign form, and rapidly terminates in recovery. *Lead* palsy alone very rarely causes death, but is usually attended by a bad general state and is of long duration and a disabling character. *Alcoholic* neuritis is more frequently fatal and is customarily associated with other organic disease due to the same cause.

The intense painful conditions that attend it and the debilitating suffering, with the tendency to mental disturbance, give to the alcoholic form a high grade of gravity. *Auto-intoxication* arising from the indole usually is the result of chronic and intractable bowel disease, and is correspondingly protracted and unmanageable. *Acute uræmia* and rapid evolution of a polyneuritis lead at once to the apprehension of respiratory and cardiac failure and an early fatal termination. On the other hand, an *insidious onset* and slow advancement imply chronic tenacity and a prolonged impairment of powers, with slow convalescence.

The gravity of a polyneuritis is in some proportion to its extent, particularly in relation to its advancement toward the trunk, implying great helplessness, and toward the pneumogastrics, implying fatal accidents from its implication. The presence of *cerebral symptoms*, such as psychoses or convulsions, and of

*spinal cord-stretch*, as shown by loss of sphincter control, add greatly to the gravity of the outlook. The general state, the powers of digestion, assimilation, and the ability to sleep have a strong bearing on the respective prospects of the patient.

**Treatment.**—The first element in the treatment of a multiple neuritis naturally is the removal of the cause. In alcoholic cases this is frequently a matter of great difficulty unless the patient can be placed under the care of a trained nurse or in a hospital. In some instances the immediate withdrawal of alcohol is attended with



FIG. 117. The upper figure represents the shape of the lower-limb pattern, and of the leather cast in correspondence to it. The dimensions are approximate, varying with each leg; *d* is the narrowing for the knee, that the leather may not press on the bone at the joint; *k* is the extension on each side for the ankle with the narrow connecting piece of leather, which is the special feature of the construction. This is 1 of 1/2 inches wide, and no longer than is needed. Affix leopardskin, adequate support to make the pull from above act down on the foot-piece on which the pull acts; at *k* and *g* are the rings for the cords; the cords are here shown only as going to the lower rings; if there is contraction of the knee, they should be carried through these rings and fastened to the other rings above the knee; if there is no contraction of the knee, it is not necessary for the leather to extend above the knee (after Green).

a great deal of physical and mental disturbance, but half-way measures usually only prolong the agony and frequently fail completely. After such a case has made decided improvement and if taken early this may promptly appear, a very little indulgence in alcohol is likely to cause the return of all former symptoms. Very moderate drinking is capable of maintaining them indefinitely.

Lead can be eliminated from the system by the use of the alkaline iodids and sulphates, but some care must be taken not to liberate the metal too rapidly, as cerebral disturbances and an increase of the paralytic features may be precipitated. They should be commenced in small but increasing doses, and aided by elimination from the skin, kidneys, and especially from the constipated bowels. Occupations giving rise to such poisoning must be given up entirely, as even when convalescence has been long established a relapse is likely to quickly follow a return to the former work.



In auto-intoxications from intestinal fermentation a restricted diet and the use of those antiseptics which tend to inhibit bacterial activity and reduce toxicity are indicated. In diphtheria the source of infection subsides with the cessation of the bacillary invasion. In the poisonings following acute infections the toxic state requires attention and is best controlled by very large doses of iron, by salol, and similar drugs. If a malarial element is present, quinine and arsenic are required. The various cachexias that are attended by the development of multiple neuritis unfortunately are rarely amenable to treatment. In tuberculosis and cancer, the measures which produce improvement in the general state benefit the neuritis. In *Beri-beri* a proper diet is probably both prophylactic and a means of cure, but the late paralytic and structural changes require the usual local methods.

The general physical condition in all cases requires attention from first to last, and one of the chief items in this connection is rest for the weakened muscles. This frequently means rest in bed. In all marked cases such rest is obligatory. In the acute cases that rapidly involve respiration and threaten the pneumogastric the greatest care must be taken to prevent pneumonia inflammation. At the same time the alimentation and support of the patient's strength frequently require the use of the esophageal tube by the way of the nose or mouth, or rectal feeding may be employed. From first to last, also, measures must be taken, especially in alcoholic cases, to prevent contractures and posture deformities. These usually are confined to the lower extremities. Foot-drop and inversion of the sole constitute the ordinary deformity, but the knees and hips are also frequently involved in a semiflexion made rigid by tedious contractures and rarely by joint-adhesions. The weight of bed-clothing serves to increase the foot-drop, and must be taken off the toes. So far as possible the feet should be kept at a right angle with the legs and the knees and hips extended. The sensitiveness of the skin and the pains in the limbs often defeat attempts to use any fixation apparatus such as light splints, but if commenced early, they can often be worn with great advantage. Pillows may be so placed as to support the feet and remove the weight of bed-covering. Passive movements in full extension and especially full dorsal flexion of the feet on the legs should be gently used several times daily.

In early stages the use of hot or cold applications has been recommended, but probably does little good, aside from slightly relieving the pain. Vesication and strong stimulation by counter-irritation must be avoided, owing to the likelihood of producing indolent ulceration. Vigorous massage and electrization that produces firm contraction of the muscles are also contraindicated at first. Gentle strokings or kneadings of the muscles that do not cause pain or distress are often very grateful to the patient, aid return circulation, and favor local nutrition. Hot baths, needle douches, Turkish and Russian baths, and rigorous measures generally should be reserved until the disease has passed its maximum.

The question of anodynes here, as in sciatica and other prolonged painful affections, is one requiring mature judgment and great conserva-

atism. The coal-tar derivatives sometimes act well and are not so objectionable as opiate and cocaine, which alone seem capable of controlling the pains in the severest cases. Chloral and bromids combined are the surest sleep-producers.

After the disease has reached its stationary period more vigorous local measures should be instituted. Massage of the weakened muscles and their exercise by galvanic electricity should be faithfully and systematically carried out once or twice daily. The wasted muscles frequently respond to static sparks before they will to the constant current, and this form of electricity, if available, will then render good service. A faradic brush or static sparks also seem to beneficially influence cutaneous anesthesia and hyperesthesia. The patient must be urged to use the limbs, and at the first possible moment to bear weight on the feet and get the heels to the floor.

In cases of long standing, when the contractures are strongly developed, stretching, under anesthesia, and the application of fixative dressings may be done, and, if this does not suffice, tenotomies are in order. In chronic cases of wrist-drop the use of splints, as described on page 207, will often be found of signal service. Here, too, courses of hot baths, especially if combined with intelligent massage, hot douches, the Scotch douche, and Turkish baths, are serviceable if the general condition of the patient does not contraindicate their use.

## CHAPTER V. HERPES ZOSTER.

HERPES ZOSTER, also known as *zoster*, *rose*, and more commonly as *shingles* (Lat. *singulus*), is an acute infectious disorder beginning with pain, soreness and burning in a given segmental skin area, followed by discrete groups of papules on a reddened and swollen base, after a few days showing turbid and purulent contents, and later brown crusts which fall by the end of the second week, leaving reddened or pigmented patches and occasionally scars. There is sometimes fever in the initial stage which with the pain subsides upon the appearance of the eruption. Secondary infection of the eruption may complicate the course and termination of the disease. The lesion is an acute hemorrhagic inflammation of the corresponding posterior root ganglia. The favorite location is on the head or face and about the trunk, but any portion of the cutaneous expanse may be affected.

**Etiology.**—The exact nature of the infection in herpes zoster is not yet fully determined, but it presents seasonal, epidemic, contagious, and self-limited peculiarities. To this infection, or the toxin therefrom arising, the posterior root ganglia show a decided susceptibility, but commonly one only is affected in a given case. Rosenow and Olschak<sup>3</sup> have cultivated an alleged specific streptococcus from the tonsil, sputum, and spinal fluid of a case of herpes, and with it produced experi-

<sup>3</sup> *Journ. Infect. Dis.*, 1916, p. 477.



mental herpes and ganglionic lesions in animals. Certain ganglia are more prone to attack than others, namely, the Gasserian and those from the third dorsal to the second lumbar spinal roots. These are in relation with the gastro-intestinal tract by sympathetic afferent filers. A similar eruption appears if the ganglion be secondarily involved in any inflammatory or destructive process, and injury to a nerve-trunk may also cause an herpetic eruption limited to its cutaneous distribution. Zoster sometimes occurs in connection with malaria,<sup>1</sup> typhoid, meningitis, pneumonia, and chicken-pox,<sup>2</sup> and after arsenical, carbonic acid gas, and intestinal poisonings. Second attacks are so rare as to lend support to the idea that immunity is afforded by the first invasion. Herpes zoster is also frequent in tabes and parietic dementia, both of which commonly involve root ganglia.

**Pathology.**—Bornasprung first found that zoster was associated with inflammatory changes in the nerve and ganglia. Read and Campbell<sup>3</sup> have placed the matter on a secure foundation. In all well-examined cases an acute inflammatory condition is found in the ganglion with bloody extravasation, destruction of cells and fibers, and inflammation of the sheath. These are followed by acute degeneration of greater or less amount and even by secondary sclerosis. In the peripheral areas degeneration appears and disappears or may terminate in sclerosis pari passu with the change in the ganglion. The nerve changes are secondary to the ganglionic invasion, but an active hemorrhagic inflammation may extend into the nerve from the ganglion. The spinal cord presents acute degeneration of the root fibers in the posterior column, appearing about the tenth day of the disease. The vesicles of the skin eruption contain a sterile serum, and no evidence of bacterial invasion is found in the neighboring profoundly inflamed lymphatics.



Fig. 128.—Herpes zoster on head in the 30th and sixth dorsal root segments.

The disease may be considered as analogous to poliomyelitis anterior acuta, and the authors quoted have proposed for it the name acute posterior poliomyelitis.

**Symptoms.**—Zoster furnishes, according to Max Joseph,<sup>4</sup> about one per cent. of all so-called skin diseases, attacks both sexes equally and mainly between the ages of fifteen and thirty years. Both sides

<sup>1</sup>Goldberb and Francis, "Jour. Amer. Med. Assoc.," April, 1918, p. 1062.

<sup>2</sup>Wardell, "N. Y. Med. Jour.," Aug. 2, 1902.

<sup>3</sup>"Trans.," Dec., 1900.

<sup>4</sup>"Phila. Med. Jour.," Oct. 25, 1902.

of the body are affected with equal frequency, but bilateral herpes zoster is extremely rare. There is an old superstition that should shingles entirely girdle the body it would be fatal. As a matter of fact, bilateral herpes zoster is usually due to serious disease of the vertebral column such as cancer or destructive Pott's disease. Epidemics of zoster occur in the spring and autumn, but sporadic cases appear with regularity throughout the year. There is a prodromal period of a few days with malaise and slight temperature. Pains, sometimes intensely neuralgic, burning, discomfort, uneasiness in varying degree, are felt in the part, and suddenly the eruption appears. Commonly, pain and temperature then subside and the eruption runs its indicated course. In exceptional cases intense localized neuralgic pains persist indefinitely, even over periods of many years, and may present paroxysms and periods suggestive of the critical attacks of tabes. Division of the posterior roots after spinal laminectomy may be required for their relief. Not infrequently, from infection of the eruption by various applications or the fingers, sloughs and even gangrene may result, ordinarily under any cleanly management the blebs dry up and to serious scarring results.

The distribution of zoster is peculiar and significant. On the head and face it follows the distribution of the three main branches of the fifth nerve very closely in some instances and rarely invades the fields of two of them in one patient. More commonly it is confined to a patch within the territory of a single branch and located at the so-called max-

imal point of the segmental area outlined by Head (see page 60). On the trunk the eruption is either massed about the maximal points of pain in the segmental areas related to the spinal cord, or, as in the case illustrated, follows precisely the limits of such area. When occurring on the extremities, the eruption follows the longitudinal lines of the cord segments. The eruption does not follow peripheral nerves and their cutaneous distribution unless arising from injuries or inflammation of such nerves.

**Diagnosis.**—The diagnosis is very readily made, only vesicular eczema and simple herpes are likely to be confounded with it. The course of the malady



Fig. 118.—Herpes zoster confined to the fifth and sixth dorsal root segments. Dorsal view.

will clear the problem as well as the anatomical relations of zoster which are not presented by eczema. Herpes occurring after arsenic and carbonic acid gas poisoning and herpes associated with pneumonia, meningitis, malaria, and typhoid present identical changes in the



ganglia, nerves, and skin.<sup>1</sup> In these forms the girdle distribution is not pronounced, and the skin eruption tends to be symmetrical, though typical zoster may be encountered in these associations.

Herpes simplex, affecting the lips and nose in coryza and gastrointestinal intoxications, and herpes genitalis are still unclassified as to the participation of the root ganglia.

**Treatment.**—The treatment seems comparatively unimportant. An antiseptic local dressing, preferably a dry one, a free action of the bowels, and the elimination of any discoverable toxic or infectious element are the indications. If the pain or burning sensation is severe a one per cent. cocaine ointment will give relief.

<sup>1</sup> Howard, "Amer. Jour. Med. Sciences," Feb., 1903.

## PART V.

# DISEASES OF THE CORD PROPER.

### CHAPTER I.

#### LOCALIZATION.

**Anatomical Considerations.**—The spinal cord reaches from the foramen magnum to the lower border of the body of the first lumbar vertebra. From its lower portion the lumbar and sacral nerve-roots extend to their several intervertebral foramina and make up the cauda equina, which occupies the dural sheath from the lumbar enlargement of the cord to the bottom of the second canal. The relations of the cord to the surface of the body and to the vertebral bodies and the spinous processes are shown in figures 120 and 121.

The cord is to be considered as made up of a number of similar and partly independent segments corresponding to the vertebral bodies, and each bearing a pair of spinal nerves. In addition it furnishes a longitudinal pathway for afferent and efferent nervous impulses. In early fetal life these cord-segments are in apposition with their corresponding vertebrae, but become gradually displaced upward as the spinal column outgrows the cord. At birth the lower end of the cord is opposite the third lumbar body. The nerve-roots, as a result of the unequal vertical growth of the cord and spine, become progressively longer from top to bottom, and in the same way the spinal-cord segment occupies a position relatively higher than its vertebral centrum. In the cervical region this amounts to the height of about one vertebral body, and in the dorsal region to about one and a half vertebral bodies. The five lumbar, five sacral, and one or two coccygeal segments are crowded into the lumbar enlargement below the upper level of the eleventh dorsal and above the second lumbar vertebral body.

In estimating the position of the various vertebral bodies we are guided by the subcutaneous tips of the spinous processes. It will be recalled that these have a downward direction in the cervical and dorsal region and overlap the body of the vertebra below. Thus the tip of the eighth dorsal spine is on a level with the ninth dorsal body. Reference to figure 121 will make this clear. It follows that the exit of the spinal root at the intervertebral foramen has only a relative relation to



the cord-segment from which it originates. Cord-lesions, are, therefore, always above the level of their spinal-nerve symptoms.

Every spinal-cord segment possesses motor, sensory, and reflex functions besides vaso-motor, visceral, and trophic activities. These are related to corresponding body-segments, which are shown in their entireness extent in figures 17 and 20. Their relation to the viscera has already been indicated (see p. 58).



Fig. 118.—The vertebral column shows the relations of the vertebrae to the corresponding spinal segments of the cord.



Fig. 119.—Showing the position of the spinal cord to the body surface.

Cross-sections of the cord show that the gray and white matter vary in proportionate area and outline at different levels (Fig. 123). The white matter is divided into numerous zones and tracts. This division is based on the embryological development of the various tracts (Fig. 124). From dissections, physiological experiments, and especially from the results of morbid processes, we have still further division of the tracts. These are shown in

figure 125 for the ascending degenerations which follow complete division of the cord, and for the descending degenerations after similar lesions.

The H-shaped cross-section of the gray matter shows numerous cell-bodies. Of these the anterior cornual groups are the best understood and contain the cell-bodies of the lower motor neuron through which motor, trophic, and vaso-motor influences are exercised over the muscles, bones, and skin. The central canal is situated in the gray commissure. The essential elements of the cord are supported by fibrous tissue which is continuous with the penetrating septa of the pia mater.

The circulation in the cord is one of its most important anatomical features. Many of the cord diseases are due to vascular lesions or infections, and both their vertical distribution and lateral outlines in transverse sections may be limited to the corresponding arterial fields.



Fig. 126.—Transverse section of cervical spinal cord, showing its anatomical subdivisions (Schuchard).

The arterial supply of the cord consists of two systems, anterior and posterior. The *anterior spinal artery*, arising within the skull by two roots from the vertebrals, extends the entire length of the cord at the anterior fissure (Fig. 127). It is reinforced by branches from the intercostal, lumbar, and sacral arteries, which follow the corresponding nerves into the spinal canal and accompany the nerve-roots to the cord. The anterior spinal artery gives off about three hundred branches, called *anterior spinal arteries*, which penetrate the anterior fissure at a right angle to the parent stem. At the commissure they enter the cord and without dividing turn toward the right or left anterior horn. At the neck of the horn the artery divides into an anterior branch to the anterior horn and a posterior branch which is distributed to the neck and to a portion of the posterior horn. A branch is also given off in a



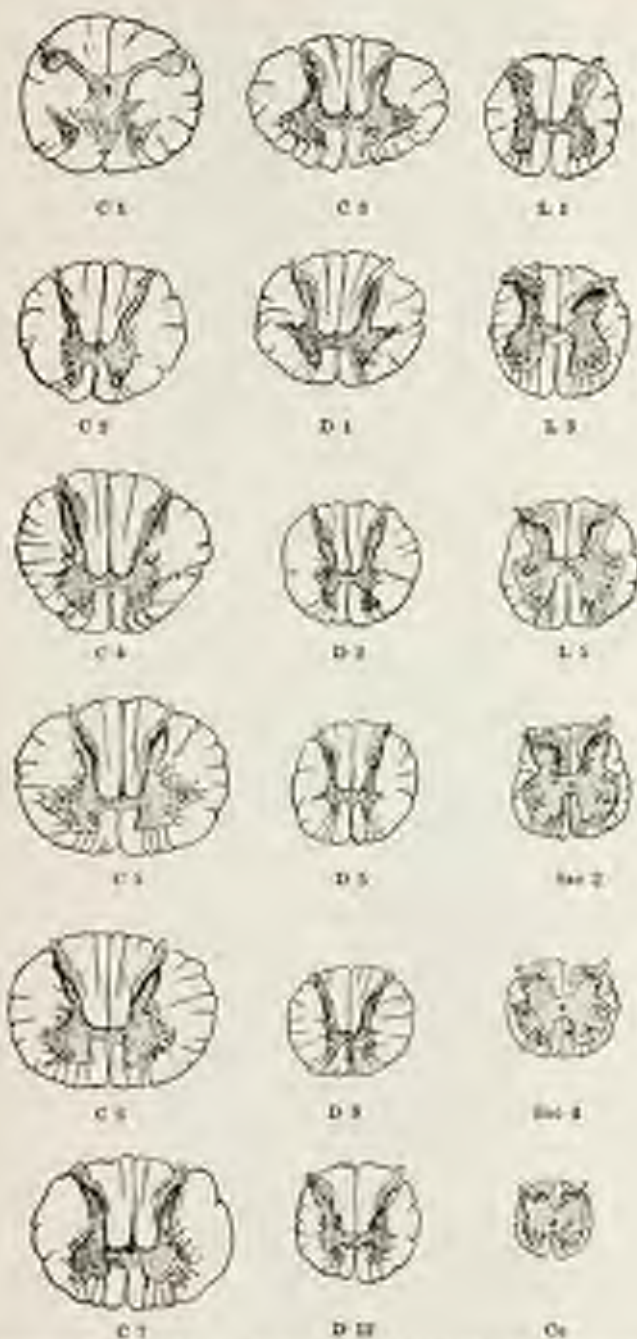


Fig. 100.—Transverse sections of the cerebellum at various levels, to show the relative variations in gray and white matter.

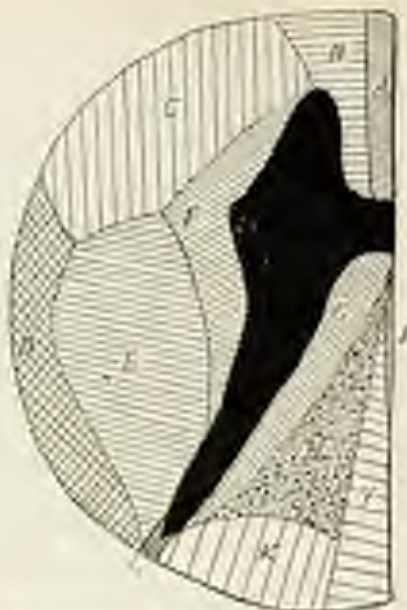


Fig. 121—Diagram of the construction of the rock based on Chodkovsky's study of Gaid development. A, first pyramidal tract; B, transverse tract; C, first of lateral tract; D, first of lateral tract; E, transverse tract; F, lateral tract; G, lateral tract; H, lateral tract; I, lateral tract; J, lateral tract; K, lateral tract.



Fig. 122—Diagram showing secondary development. A, first of the ascending tract; B, transverse tract; C, first of lateral tract; D, transverse tract; E, transverse tract; F, lateral tract; G, lateral tract; H, lateral tract; I, lateral tract; J, lateral tract; K, lateral tract.



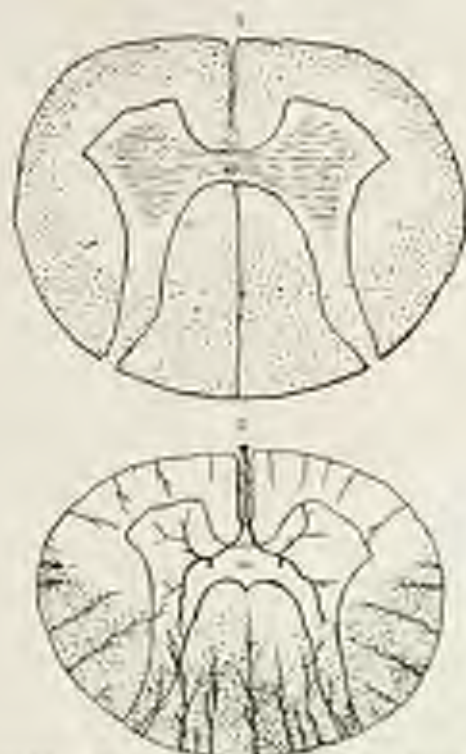


Fig. 126.—Localization of the cerebellum. Division from three arterial trunks (*Ungemach*). Part supplied only by posterior *arteria cerebri* and its branches is shaded with parallel lines. Part supplied only by the posterior *arteria cerebri* is shaded with dots. Part supplied by both *arteriae cerebri* is shaded with cross-hatching. 1. Distribution anterior and middle trunks of *arteria cerebri* and posterior *arteria cerebri* shaded after *Meyer*. Part supplied by posterior *arteria cerebri* shaded with dots. Part supplied by anterior system shaded with cross-hatching.

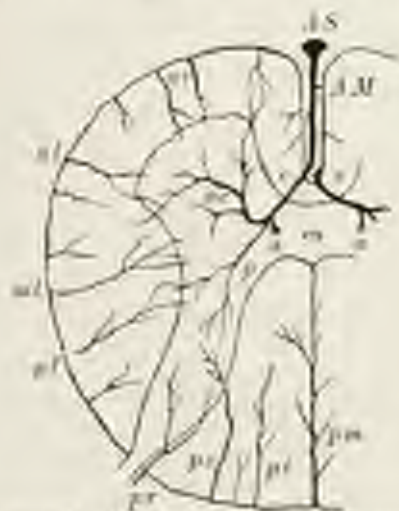


Fig. 127.—Arteries of the cerebellum. A.S. anterior superior; A.M. anterior middle; A.I. anterior inferior; M.I. middle inferior; P.I. posterior inferior; P.M. posterior middle; P.S. posterior superior. Part shaded with cross-hatching is supplied by both *arteriae cerebri*. Part shaded with dots is supplied by the posterior *arteria cerebri*. Part shaded with parallel lines is supplied by the anterior *arteria cerebri*.

vertical direction—communicating with the next similar artery, and forming a longitudinal chain in the gray matter (Fig. 128). The anterior spinal artery, from its lateral ramifications on the surface of the cord, also supplies the white matter in the periphery of the ventral half of the cord.

The posterior arterial system of the cord supplies its dorsal half. The two *posterior spinal arteries* arise separately from the *vertebrals* and, coursing around the *nodulla*, extend the entire length of the cord just outside the posterior nerve-roots. Like the anterior spinal artery, they receive numerous supply branches from the intercostal and lumbar arteries. They give off *anastomotic branches* which unite

with those of the anterior system on the surface of the cord, and also present a chain of anastomosing branches on the posterior median line of the cord. From these branches small twigs penetrate the cord supplying the gray matter of a portion of the posterior horn and the posterior half of the white matter of the cord (Figs. 127 and 128). The arterial twigs entering the cord are of the terminal variety, and therefore do not anastomose. The territories of the two systems are not entirely independent, as the borders of their irrigation fields overlap and the adjacent white and gray matter receive arteries from both sources. Three arterial districts are thus constituted: (1) that supplied only by the anterior system; (2) that supplied only by the posterior system, and (3) that irrigated by both systems (Fig. 126).

It will be apparent from these facts that arterial disease may induce lesions in the posterior half of the cord, or in the anterior half. Further, the infection or obliteration of a single anterior median artery will practically destroy the corresponding anterior horn.

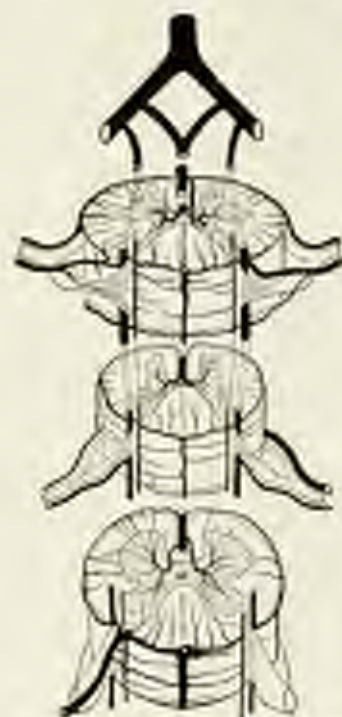


Fig. 128.—Continuation of those circulating in cord separately and in vertical direction (Erdmann).

The circulation in the cord, as a whole, presents some mechanical conditions that are significant. The spinal arteries are the largest of their size in the body, and, owing to their course, are not subject to the direct impact of cardiac impulses. In this respect they are very different from those of the brain. Arterial pressure is also slight, and the venous outlet into the plexuses about the dura is not as advantageous one in the erect position. Gravity impedes the return circulation as well as the supply. This is most marked at the lower end of the cord. The long course the lumbar and sacral arteries have to pursue in an upward direction along the corresponding nerve-roots in the cauda still further reduces the circulatory qualifications of this end of the cord.



These mechanical disadvantages may perhaps partly explain the liability of the cord to gas embolism in caisson disease and the localization of various infections.

The arteries in the cord are provided with perivascular lymph-sheaths, which are continuous with the arachnoid meningeal spaces. Veins constantly accompany the arterial branches and empty into posterior and anterior plexiform venous chains on the surface of the cord, discharging in turn into the extradural plexuses. Regurgitation from the extradural veins is probably impossible.

The posterior roots bear a ganglionic enlargement situated just outside the dura, except on the lowest pairs, where it is within the sheath. Its function is trophic for the afferent tracts in the cord.

**Vertical Localization of a Cord-lesion.**—Cord-lesions are irritative or destructive and are manifested by corresponding symptoms in the periphery to which the injured cord-segments are anatomically related and to which their peripheral nerves are distributed. Irritative lesions are marked by sensory exaggeration, such as hyperesthesia and pain, by cramps and increased reflexes. Anesthesia, paralysis, and abolished reflexes result from destruction of a cord-segment. Trophic loss and vasomotor paralysis may also ensue, but become apparent at a later date. The symptoms of a cord-lesion embrace: (1) Those due to the derangement of the injured segment; (2) those resulting from disturbances in the conduction attributes of the cord, and (3) those arising from the secondary degenerations in the cord. These are variously combined, depending upon the extent of the lesion in the cross-section of the cord and its age.

**Motor Symptoms.**—A completely disabling injury falling upon any cord-segment produces paralysis in the muscles supplied by that segment. It will be noticed, in the following table, that muscular actions are functionally grouped in the cord and extend in a vertical direction through several segments. Single muscles are, therefore, practically never alone paralyzed by spinal lesions. Such a circumscribed palsy, or one confined to a group of muscles supplied by a single nerve-trunk, at once declares the peripheral character of the lesion. Not only are the muscles supplied by the injured segment paralyzed, but all muscles represented in the cord below the lesion are withdrawn from voluntary control through the division of their motor paths in the upper neurone, which traverse the cross-lesion by way of the pyramidal tracts. A paraplegia is thus induced.

If the lesion is not a destructive one, but irritative in its effects, spasms and rigidity may be present. These are usually attended by clonus in the large muscles whose tendons pass over joints; they are present in all the levels below the upper limit of the lesion, but more especially in the lower limbs.

In late cases of partial cross-lesions the rigidity and *spastic posture* are well developed. The lower limbs are held rigidly extended or, less frequently, sharply flexed. Any attempt to move them causes clonic trembling, which may finally end in a sharp pulling up of the leg with a "jack-knife"-like jerk, or the thighs may be held so rigidly by the muscles of the pelvic girdle that if one limb is raised from the bed by the foot the other follows with it. *Contractures* finally develop: (1) By the paralysis of one group of muscles and the unopposed over-

action of antagonists; or (2) by the overirritation of the motor mechanism of certain muscles or muscle-groups; or (3) by structural changes in the muscles, causing a tendinofibrosis, with its characteristic retraction. Only when dealing with the third form is function permanently useful, as in the other varieties the activity of the muscular masses promptly reproduces the conditions sought to be relieved. In the structural variety the dislocated joint usually has a certain limited range of free motion, but is then abruptly stopped by the retracted, unyielding tendons, which stand out prominently. On the other hand, the muscular contractures, due to overstimulation in which fibroid changes have not taken place, usually yield slowly or, perhaps, jerkily to efforts tending to extend them.

MOTOR AND REFLEX FUNCTIONS OF THE SPINAL-CORD EXEMESIS  
(AFTER STARR AND THOMSON.)

SEGMENT.	MUSCLES.	REFLEXES.
Cervical	Sternomastoid.	
1-2	Trapezius.	
	Scaleni.	
	Small rotators of head.	
	Diaphragm.	
	Lev. ang. sup.	Instinctive or pupal by (fronting side of neck, 4-5 cervical.
	Blasphonds.	
4	Spinati.	
	Deltoid.	
	Scapular ang.	Scapular reflexes, 5 C-1 D.
5	Dorsus.	Scapular ang., 5 C.
	Scapular. brach.	
	Serratus. ang.	
	Pectoralis (lat.)	
	Torso. extensor.	
6	Proneus.	
	Brachialis ant.	
	Triceps.	
	Long. extensors of wrist and fingers.	
7	Flexor.	
	Peroneus (lateral).	
	Latiss. dorsi.	
	Torso. quad.	
	Long. flexors, wrist and fingers.	
8	Extensors of thumb.	
9	Extensors of thumb.	
10-12	Extensors of thumb.	
11-12	Extensors of thumb.	
13-14	Extensors of thumb.	
15-16	Extensors of thumb.	
17-18	Extensors of thumb.	
19-20	Extensors of thumb.	
21-22	Extensors of thumb.	
23-24	Extensors of thumb.	
25-26	Extensors of thumb.	
27-28	Extensors of thumb.	
29-30	Extensors of thumb.	
31-32	Extensors of thumb.	
33-34	Extensors of thumb.	
35-36	Extensors of thumb.	
37-38	Extensors of thumb.	
39-40	Extensors of thumb.	
41-42	Extensors of thumb.	
43-44	Extensors of thumb.	
45-46	Extensors of thumb.	
47-48	Extensors of thumb.	
49-50	Extensors of thumb.	
51-52	Extensors of thumb.	
53-54	Extensors of thumb.	
55-56	Extensors of thumb.	
57-58	Extensors of thumb.	
59-60	Extensors of thumb.	
61-62	Extensors of thumb.	
63-64	Extensors of thumb.	
65-66	Extensors of thumb.	
67-68	Extensors of thumb.	
69-70	Extensors of thumb.	
71-72	Extensors of thumb.	
73-74	Extensors of thumb.	
75-76	Extensors of thumb.	
77-78	Extensors of thumb.	
79-80	Extensors of thumb.	
81-82	Extensors of thumb.	
83-84	Extensors of thumb.	
85-86	Extensors of thumb.	
87-88	Extensors of thumb.	
89-90	Extensors of thumb.	
91-92	Extensors of thumb.	
93-94	Extensors of thumb.	
95-96	Extensors of thumb.	
97-98	Extensors of thumb.	
99-100	Extensors of thumb.	



**Sensory Symptoms.**—A cross-lesion destroys cutaneous sensation in the segmental area corresponding to the lesion and in all parts below, the first directly, the second by interruption of conduction. The distribution of *anesthesia* is practically the best guide to the upper level of the lesion. In the diagrams shown on pages 56 and 59 it will be seen that the cutaneous areas of the skin do not correspond exactly to the cutaneous distribution of the spinal nerves. This variation will often serve to differentiate between a cord-lesion and one of the nerve-roots. Root-lesions cause areas of sensory disturbances that coincide with the cutaneous distribution of the nerves arising from them. In the trunk, for instance, division of the cord produces an *anesthesia* whose upper level is practically in a horizontal plane, while root-lesion *anesthesia* or *hyperalgesia* follows the intercostal nerves and spaces. The territories supplied by the intercostal nerves overlap decidedly, so that destruction of a single nerve may manifest no sensory loss. It requires that at least two should be divided to produce an anesthetic patch or belt.

The upper border of a spinal lesion is usually further indicated by a zone of *hyperesthesia* due to the irritant action of the lesion on sensory paths within the cord or upon the posterior nerve-roots. This is described by the patient as a band or girdle-like sensation about the trunk and as tightness in the limbs. Its vertical extent corresponds to the irritating influence, but rarely does it exceed two segmental areas. A light touch in this hypersensitive zone causes a feeling as of pins and needles, of pricking, of burning, of tingling, or of a thrill, and is not a pure exaggeration of sensation, but a perversion of it. Very severe pain is usually absent in pure cord lesions, but the girdling sensation is often described as painful; and if the posterior roots are injured, as in Pott's disease or through meningeal inflammation, the pains are intense, and often darting in character. *Spontaneous sensations* are frequent, and patients often attempt to describe peculiar feelings of an unnatural sort which they may locate in areas that are absolutely insensitive to external stimulation. They may be caused by the irritation of the conduction tracts at the upper level of the lesion, and are then referred to the peripheral sites from which they would naturally arise.

In lesions of lesser degree sensation may not be much affected, even when motion is abolished, or the general sense of touch may be broken up so that temperature and painful impressions are not recognized. Tactile impressions, in the same way, may fail to arouse the sensorium when more energetic and painful impressions still traverse the injured pathways.

The reflexes furnish very valuable evidence not only as to the upper level of the cord lesion, but sometimes as to its vertical extent. The cord lesion that destroys the portion of a reflex arc within the cord obliterates that reflex but does not abolish the reflexes below its own level unless the cord is entirely and completely divided. It is now fairly well established that complete division of the cord in man extinguishes all cord reflexes below that level.<sup>1</sup> On the contrary, if the division is incomplete, the lower reflexes may at first be enfeebled, but within a few weeks show much exaggeration. In a case showing such increased reflexes the absence of one or more at a given level points to the diseased portion of

<sup>1</sup> Osler, "Brain," Spring, 1904.

the cord. Again, the upper level of abolished reflexes usually coincides with that of anesthesia, and both localize the lesion. In other cases the extension and invasion of cord disease may be traced by the successive disappearance of spinal reflexes; its recession, by their reappearance.

**Trophic Conditions.**—As the trophic centers for muscles correspond with their motor spinal centers in the anterior horns, a lesion which destroys this portion of the gray matter of a cord's segment induces atrophy in the related muscles. This atrophy, like the palsy arising from a cord lesion, has a functional distribution that depends upon the association of muscular representation in the cord. By reference to the table, page 348, it is evident that all of a large muscle need not be involved, and that groups of muscles innervated by different nerve trunks, but centrally associated, may be thus selected. The extent of the wasting is limited by the vertical dimensions of the lesion. The muscles innervated from the cord above and below the destructive process are spared, and retain their nutritional supply and their electrical responses. The wasted muscles lose tone early, and shortly thereafter present the electrical reaction of degeneration. Atrophic loss is best seen in the muscles of the extremities; next in those of the shoulder and pelvic girdles. In the thoracic and abdominal regions the wasting is only perceptible when several adjacent cord segments are diseased.

Slight atrophic disturbances appear in the skin, analogous to those in neuritis and confined to the area related to the injured cord segments. In the case of irritative lesions, such as acute myelitis and cord hemorrhage, the dystrophic condition may be acute and intense. Under the influence of comparatively slight pressure or other superficial irritation, herpetoid eruptions and acute bedsores form in a few hours. These occur over the scapula, heels, malleoli, and trochanters by preference, but may occur wherever the bones are subcutaneous and the trophic control disturbed. The use of counterirritation in the mildest form, and even of frictions with the hand, may provoke them. Hot bottles that would otherwise cause no injury may, under these circumstances, induce most serious local effects and deep destruction of tissue.

Vasomotor disturbance in some degree is usually present, and consists, ordinarily, at first of a tendency to vascular dilatation and increased warmth. The so-called *brûle étouffé* is easily provoked below the lesion. In cases of long standing the skin is livid and cold, frequently with increased perspiration. Lesions in the cervical region often cause flushing and perspiration on the side of the neck and face, and may reduce the heart-beats to forty or even to twenty a minute. Dorsal lesions, on the other hand, are sometimes attended by a persistently rapid pulse. These vasomotor disturbances are frequently attended by an increase of body temperature in lesions in the upper dorsal and lower cervical regions, but it is often difficult to exclude pyrexia, due to the infectious nature of the disease or to intestinal or vesical disturbance resulting from it.

Visceral symptoms are usually not pronounced, but the secretion in the alimentary canal and its muscular activity are frequently disturbed. Constipation is the rule, and fermentation of the stomach and intestinal contents, with gaseous disturbance and great abdominal distention, is very



emission. Vomiting and difficulty in swallowing occur in lesions of the cervical cord. The anal and vesical sphincters are usually disturbed. When the lesion involves their reflex centers in the lumbar cord, complete relaxation and incontinence ensue; but if the lesion is above their spinal centers, voluntary control alone is lost. They then act automatically, and the corresponding viscera are unconsciously evacuated at intervals. The examining finger readily provokes the anal sphincteric contraction in this condition, which is not the case if its center is destroyed. There is a tendency to fecal and urinary retention, dependent, in part, upon the lack of power in the abdominal muscles. In the case of the bladder this leads to distention by increased residuum and weakening of the detrusor, and the dilatation of the viscus may become extreme. The result is usually a cystitis, which is often precipitated by the use of a septic catheter. Damage to the kidneys arises both from the back-pressure of urine and the propagation of inflammation up the ureters. The table of symptoms in disabling but not absolutely destructive transverse cord lesions (see pages 353-359) is based upon the exhaustive work of Wichman.<sup>3</sup> If the entire cross-section is absolutely destroyed, the symptomatology is the same excepting that there is complete absence of muscle reflexes below the lesion.

**Horizontal Localization of a Cord Lesion.**—Many spinal cord lesions are more or less circumscribed in transverse area, and give rise to widely different symptoms as they affect the various physiological divisions of the cord. Some cord diseases are symmetrical, and both sides of the transverse section show identical conditions. Others are unilateral, and the two sides show different states, not only at the level of the lesion, but also in the levels affected by the secondary ascending and descending degenerations.

Lesions of the *pyramidal tracts* produce motor paralysis below the level affected and induce a spastic condition in the paralyzed area, marked by increased muscle tonus, exaggerated reflexes, rigidity, and contractures. These tracts degenerate downward.

Disease of the *posterior columns* is marked by sensory disturbance, especially of those elements of touch related to pressure and to the muscle and joint sensations. Ataxia results. The temperature and pain sensations are also usually diminished, and all forms of cutaneous impressions are delayed in reaching the brain. The muscle reflexes, especially the deep tendon reflexes, are abolished or greatly diminished. An upward degeneration in the postero-lateral column ensues.

When the *anterior horn* is affected motor paralysis occurs, but only in the muscles which are supplied by the large cells actually involved in the morbid focus. The muscles also atrophy. If the process is acute, paralysis takes place at once and atrophy gradually develops. In very insidious lesions paresis and atrophy develop at an equal pace, and fibrillary twitchings are usually present. Reflexes are abolished by lesions of the anterior horns and vasomotor paralysis is induced in the field related to the cerebral disease. Degeneration descends the lower neurons arising from the diseased portion of the cord, and the reaction of degeneration is presented in nerve and muscle.

<sup>3</sup> "Die Rückenmarksnerven und ihre Segmentzuge," Berlin, 1906.

Disease of the *posterior horns* is marked by sensory disturbances or *anesthesia* in a given area, such as follows a lesion of the posterior column.

Lesions of the *posterior roots* cause anesthesia if complete; hyperaesthesia and radiating pain if irritative.

Lesions of the *anterior roots* produce the same results as lesions of the anterior horns.

Lesions arising *within* or *immediately about* the *central canal*, as in *syringomyelia*, produce a peculiar dissociation of touch sensations, so that painful and thermal impressions are not properly recognized while tactile or contact impressions remain practically unaffected. Joint dys trophies are often added.

A lesion that divides *one lateral half* of the cord gives rise to the *Brown-Séquard syndrome* (see p. 56).

In many cord diseases two or more physiological divisions are symmetrically affected. In *anotrophic lateral sclerosis* we have, added to the rigidity, myotatic irritability, and contractures that mark disease of the lateral tracts, a progressive muscular atrophy that is due to the lesion of the anterior horns. *Ataxic paraplegia* is marked by symptoms in both the lateral tracts and the posterior columns, and we find ataxia and rigidity with weakness variously combined. The following table roughly shows the relation of the various cord diseases to the physiological division of the cord. Those which are marked by lesions confined principally to given tracts in the spinal cord are denominated *system diseases*, as distinguished from indiscriminate lesions. As will appear in the consideration of individual diseases, some of these cord lesions, as in *locomotor ataxia*, are only a part of the morbid findings.

Organic diseases of the cord.	Indiscriminate lesions.	Hemorrhage.		
		Myelitis.		
	System diseases.	Lesions of single tracts.	Anterior cornu.	Polio-myelitis acuta.
			Lateral tract.	Progressive muscular atrophy.
		Posterior columns.	Primary lateral sclerosis.	
			Little's disease.	
		Combined lesions.	Lateral and posterior columns.	Lathyrism.
			Anterior cornu and lateral tract.	Locomotor ataxia.
		Lateral and posterior columns.	Ergotism.	
			Anterior cornu and lateral tract.	Atrophic lateral sclerosis.
Lateral and posterior columns.	Ataxic paraplegia.			
	Friedreich's ataxia.			
Lateral and posterior columns.	Hereditary ataxia.			
	Pellagra.			
Lateral and posterior columns.	Effects of cross lesions.			





TABLE OF CONTENTS: I. CLASS-LOCATIONS IN THE CORDON - (Continued)

[illegible]





[illegible]

Anatomical Description	Myoanatomy		Functional Aspects		Associated Posture/Action
	Primary Muscles	Secondary Muscles	Primary Action	Secondary Action	
III. Lumbar	Muscles of torso, bladder, and genitalia. Forward rotation of hip. Forward rotation of thigh. Forward rotation of knee. Forward rotation of foot. Forward rotation of toe. Forward rotation of heel. Forward rotation of foot. Forward rotation of toe.	Extensor digitorum. Extensor indicis. Extensor carpi ulnaris. Extensor carpi radialis. Extensor pollicis longus. Extensor pollicis brevis. Extensor pollicis longus. Extensor pollicis brevis. Extensor pollicis longus. Extensor pollicis brevis.	Extension of leg at hip joint. Flexion of leg at hip joint. Flexion of leg at knee joint. Flexion of leg at ankle joint.	Abduction of leg at hip joint. Adduction of leg at hip joint. Adduction of leg at knee joint. Adduction of leg at ankle joint.	Abduction of leg at hip joint. Adduction of leg at hip joint. Adduction of leg at knee joint. Adduction of leg at ankle joint.
IV. Lumbar	Forward rotation of hip. Forward rotation of thigh. Forward rotation of knee. Forward rotation of foot. Forward rotation of toe. Forward rotation of heel. Forward rotation of foot. Forward rotation of toe.	Extensor digitorum. Extensor indicis. Extensor carpi ulnaris. Extensor carpi radialis. Extensor pollicis longus. Extensor pollicis brevis. Extensor pollicis longus. Extensor pollicis brevis. Extensor pollicis longus. Extensor pollicis brevis.	Extension of leg at hip joint. Flexion of leg at hip joint. Flexion of leg at knee joint. Flexion of leg at ankle joint.	Abduction of leg at hip joint. Adduction of leg at hip joint. Adduction of leg at knee joint. Adduction of leg at ankle joint.	Abduction of leg at hip joint. Adduction of leg at hip joint. Adduction of leg at knee joint. Adduction of leg at ankle joint.
V. Lumbar	Forward rotation of hip. Forward rotation of thigh. Forward rotation of knee. Forward rotation of foot. Forward rotation of toe. Forward rotation of heel. Forward rotation of foot. Forward rotation of toe.	Extensor digitorum. Extensor indicis. Extensor carpi ulnaris. Extensor carpi radialis. Extensor pollicis longus. Extensor pollicis brevis. Extensor pollicis longus. Extensor pollicis brevis. Extensor pollicis longus. Extensor pollicis brevis.	Extension of leg at hip joint. Flexion of leg at hip joint. Flexion of leg at knee joint. Flexion of leg at ankle joint.	Abduction of leg at hip joint. Adduction of leg at hip joint. Adduction of leg at knee joint. Adduction of leg at ankle joint.	Abduction of leg at hip joint. Adduction of leg at hip joint. Adduction of leg at knee joint. Adduction of leg at ankle joint.



<p>333-111— Dorsal.</p>	<p>Paralysis of lower extremities, and glans region.</p> <p>Paralysis of umbilical and dorsal regions gradually added at the side at the lower extremity.</p>			<p>Paralysis of muscles of respiratory system disappears gradually and dyspnea.</p>	<p>In complete lesions, respiratory system gradually paralyzed, and the respiratory apparatus is gradually paralyzed. A.B. below lung in complete paralysis of respiratory system.</p>	<p>Increased in the complete lesions.</p>	
<p>333-112— Dorsal.</p>	<p>As in 111. Dorsal.</p>			<p>As in 111. Dorsal.</p>	<p>As in 111. Dorsal.</p>	<p>As in 111. Dorsal.</p>	
<p>333-113— Dorsal.</p>	<p>A.B. muscles of back and lower extremities.</p>	<p>Paralysis of Respiratory system of the 11th, 12th, 13th and 14th segments.</p> <p>Paralysis of the 15th, 16th, 17th and 18th segments.</p> <p>Paralysis of the 19th, 20th, 21st and 22nd segments.</p>	<p>Weakness in the 11th and 12th segments.</p> <p>Paralysis of the 13th and 14th segments.</p>	<p>As in 111. Dorsal.</p>	<p>As in 111. Dorsal.</p>	<p>As in 111. Dorsal.</p>	

TABLE OF SYMPTOMS IN CROCODILES OF THE GENUS *CROCODRUS* (Continued)[illegible]



[illegible]

**Lesions of the Cauda Equina.**—The descending roots of the lumbar, the sacral, and the coccygeal nerves make up the cauda, which occupies the dural sheath below the upper level of the second lumbar vertebra—a distance of about ten inches. As the roots are given off in lateral pairs, the upper ones are external and soonest pass into the vertebral foramina. Those that are longest, therefore, are situated toward the middle line. It is evident that a lesion involving the dural contents below the *conus medullaris* will produce a root lesion. This may be partial or complete, and varies according to the level which it occupies and the roots actually involved. The longer roots are usually most affected, even when the lesion is so placed as to embrace all the roots of the cauda extending below the *conus*. As a consequence, the nerves arising from the lowest cord segments and distributed to the lowest body levels are the ones commonly disturbed. The paralysis, anesthesia, abolition of reflex action, and atrophy that result from a complete root division correspond to the peripheral distribution of the injured nerve roots. These peripheral distribution areas correspond somewhat to the segmental cord areas, but show a marked tendency to follow the distribution of the nerve trunks. (See Figs. 15 and 16.) Both sides of the body are usually affected, but uniform symmetry is the exception, while in cord lesions it is the rule.



Fig. 12b.—Area of anesthesia in a lesion of the cauda, affecting all the sacral roots.

The lowest portion of the cord, known as the *conus medullaris*, comprises the third, fourth and fifth sacral, and the coccygeal segments. It contains the reflex centers for urination, defecation, ejaculation, and the anal sphincter. Lesions of the *conus* and the cauda present so many common points that the distinction is often very difficult. After L. R. Müller, Bruns, Cassirer, and others we may make the following differential enumeration:

The *caudal lesion* is: (1) Of slow onset except in traumatic cases. (2) It is attended by lancinating pains and painful paroxysms in the bladder, perineal, and sciatic regions. (3) Anesthesias embracing all modalities of sensation are of gradual and late development and rarely of accurately symmetrical distribution. (4) There is an absence of spasms, rigidity, fibrillation, and increased reflexes. The condition is one of flaccidity; the heel, plantar, and anal reflexes are diminished or abolished, the knee-jerk is retained, there is muscular wasting in the legs, and the reaction of degeneration in severe or advanced cases. (5) A gradually advancing paresis follows the sensory symptoms. (6) At the same time weakness of bladder, bowel, and sexual power develop. (7) There is frequently tenderness over the second lumbar spine and percussion tenderness over

The *caudal lesion* is: (1) Of slow onset except in traumatic cases. (2) It is attended by lancinating pains and painful paroxysms in the bladder, perineal, and sciatic regions. (3) Anesthesias embracing all modalities of sensation are of gradual and late development and rarely of accurately symmetrical distribution. (4) There is an absence of spasms, rigidity, fibrillation, and increased reflexes. The condition is one of flaccidity; the heel, plantar, and anal reflexes are diminished or abolished, the knee-jerk is retained, there is muscular wasting in the legs, and the reaction of degeneration in severe or advanced cases. (5) A gradually advancing paresis follows the sensory symptoms. (6) At the same time weakness of bladder, bowel, and sexual power develop. (7) There is frequently tenderness over the second lumbar spine and percussion tenderness over



the sacrum and lumbar region, where occasionally bony deformities can be detected by examination, the x-ray, etc. (8) Remissions are frequent. Schlesinger<sup>2</sup> calls attention to a painful stretching symptom when the patient is in the sitting posture. It appears to be due to the tension put upon the caudal roots by this position. The pain is referred to the rectum, bladder, genitals, perineum, or even down the thighs; it is generally intense and persistent.

The *causa lésion* is: (1) Usually of abrupt onset. (2) Pain is not a marked symptom and may be quite absent. (3) Anesthetics of perineal, gluteal, and pudendal regions are a common early symptom, of marked symmetrical distribution, and frequently of the dissociation type; testicular sensitiveness is retained. (4) Motor irritation, such as fibrillations, cramps, and rigidities, are common, but the tendon reflexes in the lower extremities are not much modified. (5) There is early weakness of bladder and bowel control with sphincteric relaxation and loss of erections. (6) Both motor and sensory features are of early appearance and advance in parallel; remissions are infrequent.

The usual lesions which affect the cauda are vertebral fractures and dislocations, new growths, penetrating wounds, and hemorrhage. In tabes and multiple neuritis, which are sometimes confounded with caudal disease, symptoms are present at higher levels, as in the upper extremities and in the pupillary reflexes. Injury to the plexuses within the pelvis usually—at least, at first—produces unilateral symptoms.

<sup>1</sup>"*Neurolog. Centralblatt*," July 1, 1915.

## CHAPTER II.

## INDISCRIMINATE CORD LESIONS.

## TRAUMATIC LESIONS OF THE CORD SUBSTANCE.

THE spinal cord is sometimes reached by penetrating wounds made by knife, bullet, or other foreign object. It is injured more frequently by the displacement of vertebrae, and this is almost invariably attended by fracture. With the surgical conditions we shall not deal. It is to be noted that vertebral fracture-dislocations are frequently devoid of any external signs of displacement, even when it is found postmortem that the vertebral bodies have been so completely displaced as to actually shear the cord in two. Restitution to a practically normal position may occur at once, either spontaneously or due to the lifting efforts of those who first attend the injured person. These cases all furnish a history of trauma, and usually present local evidence of it. The question that arises regards the location and extent of the lesion in the cord. This must be determined by an application of the considerations set forth in the foregoing chapter.

The prognosis in a case of actual injury to the cord substance is always grave and generally fatal. If the lesion cause complete division, there can be no hope for voluntary control or sensory improvement below its level. Only in very slight injuries from dislocation of the vertebrae or from pressure due to meningeal hemorrhage can much be expected. In such a case complete paralysis may pass away; but when the reflexes are completely abolished in the paralytic area after the first week, little improvement need be expected. Some degree of disability always persists, and commonly bedsores, cystitis, kidney and pulmonary complications carry off the patient after a lingering helplessness.

## HEMORRHAGE INTO THE CORD (HEMATOMYELIA).

Hemorrhage into the spinal cord is not an extremely rare accident. It occurs under a variety of circumstances, and is punctate and multiple or single and more or less extensive. There is also a so-called perforating form simply due to the tendency of the extravasated blood to follow the lines of least resistance in the longitudinal direction of the cord.

**Etiology.**—Hemorrhage into the cord, like spinal meningeal hemorrhage, with which it is frequently associated, may follow severe spinal concussions and violent wrenchings of the back. Excessive muscular effort, as in lifting, has caused it, and Gowers reports this accident following repeated colics. A focus of myelitic softening is frequently the seat of hemorrhage, and hemorrhage, in turn, is followed by a zone of myelitic softening. It is often difficult to tell which process was the initial one. In some cases ofisson disease, or divers' palsy, hemorrhage has been found due to the too sudden reduction of the high atmospheric pressure under which such work is done. Continued convulsions, as in status epilepticus, intense chorea, tetanus, and asphyxia, may produce punctate hemorrhages and hemorrhagic infiltration, especially in the



gray matter of the cord. Purpura and sudden cessation of lobular hemorrhages, such as that from piles and menstruation, may provoke a cord-apoplexy. A dilated central canal and teratological defects and fissures in the cord predispose to it. Changes in the arterial coats are much less active factors than is cerebral hemorrhagic apoplexy, as atheroma and miliary aneurysmal dilatations are of the greatest rarity in the cord. The arterial pressure conditions and the direct cardiac impulse that play so large a part in brain-lesions are also absent.

**Morbid Anatomy.**—In the punctate and infiltrating variety the cord may merely appear slightly reddened, or small, pinhead clots may be found, particularly in the gray matter. They usually first occupy the perivascular spaces and, aside from traumatic cases, are secondary to myelitic softening. The appearance varies with the size of the extravasation. The large single or focal hemorrhage also finds its usual seat in the gray matter, less commonly in the central canal or in a congenital fissure of the cord. It is usually ovoid in shape, with the long axis vertical, and may perforate the yielding gray substance of the cord for several inches in extreme cases. It causes a fusiform, dark-colored swelling of the cord. The blood usually comes from the larger median arteries or from those entering the anterior horn by the anterior roots, and favors the gray matter as a situation. In rare instances the blood may break through the white columns and even appear to a slight extent in the membranes. Meningeal



Fig. 102.—Hemorrhage into the gray matter of the cervical part below the center for the triceps and supinator longus; paralysis and atrophy of the triceps and supinator (cf. wrist) and biceps.

clots are also commonly present in traumatic cases. Around the clot, after a few days, the cord shows a zone of softening. In cases of long standing the clot may undergo changes similar to those in the brain, and a hemorrhagic cyst remains, with ascending and descending degenerations corresponding to the location of the lesion in the cross-section. A wide area of myelitis, containing a comparatively small and recent clot or hemorrhage into a glomerosus growth, may be encountered as secondary accidents.

**Symptoms.**—A primary focal hemorrhage into the cord is of rapid, usually of sudden onset. The infiltration sort, being almost always secondary, may be preceded by sensory and motor symptoms for hours, days, and even weeks. This is also true of the larger clot that forms in a softened myelitic territory, producing sudden exacerbation of the usually preexistent paralytic symptoms. The earliest symptom is commonly one of severe pains radiating in the body-segments corresponding to the hemorrhagic focus. These are due, presumably, to the pressure or laceration of the sensory tracts in or near the posterior commissure. Darning pains occur in the limbs, girdling pains in the trunk that may

be mistaken for *angina pectoris* and intestinal or vesical colic. Very shortly—that is, in the course of a few minutes or an hour—after an accident has occurred, and the patient has perhaps walked a short distance, paraplegic symptoms appear. These may be partially unilateral at first. The legs weaken, the patient gradually or quickly sinks down, and usually the motor loss is promptly established at its maximum. The condition that next develops depends on the location, extent, and size of the clot, and the amount of pressure it brings to bear on the conduction tracts. Usually there is some improvement, owing to the subsidence of the pressure and of the shock or insult to the adjacent portions of the cord. The development after a day or two of myelitic softening or myelitis, with elevation of the body-heat, usually again emphasizes the paraplegia, and thereafter the course of the disease and its treatment is that of myelitis. The bladder and anal sphincters are commonly immediately relaxed. All the tendon reflexes are diminished at first, but after a week or ten days they begin to increase, and comport themselves as in the spastic state that follows an incomplete cross-lesion of the cord. Early in the attack spasms and tonic convulsions in the muscles supplied by the affected segments and those below the lesion are frequently met with.

The diagnosis is often difficult and it is frequently impossible to exclude a meningeal hemorrhage. Prevailing sensory, motor, and temperature disturbances indicate a primary myelitis. Only in those cases where the temperature is normal, the onset abrupt, and the pain of the segmental variety, can a definite diagnosis be ventured.

The prognosis depends upon the location of the lesion. Hemorrhage into the cord in the upper cervical region is almost certainly fatal, and is worse in the cervical and lumbar enlargements than in the dorsal region. The secondary myelitis may extend upward and cause death, or downward and involve the lower levels. Only when sensation improves, motor gain is apparent, and febrile disturbance is passed, is the patient safe. Some lasting local paralysis, wasting, and trophic disturbances result, and more or less permanent spasticity remains. Too often bedsores, cystitis, sepsis, or other complications carry off the patient.

**Treatment.**—The immediate treatment of the hemorrhage consists of measures to check it. The patient should be placed face downward with the spine elevated as much as possible, and applications of ice to the loins made over the length of the cord. Absolute quiet and the determination of blood to the surface, intestinal tract, and extremities should be favored. Surgical exploration in selected cases is strongly indicated, and longitudinal incisions into the cord after the manner of Reginald Allen should be employed.

#### THROMBOTIC SOFTENING OF THE CORD.

Thrombosis of the arteries of the spinal cord undoubtedly occurs frequently. The anatomical features of the circulation in the spinal cord render this accident a very likely one. The long course the arterial supply traverses, especially in the lumbar cord, produces a sluggishness of its current that favors the deposition of fibrin if at the same time the arterial wall is nutritionally disturbed. As the spinal arteries after ex-



tering the cord are of the terminal variety, it is evident that their obliteration will result in the softening of their irrigation fields. This result has ordinarily been confounded with myelitis and clinically presents the same picture. Gowers denies its occurrence, as does Strümpell, but in syphilitic cases this mechanism is demonstrated by such cases as are described by Williamson<sup>1</sup> and those of Schmaus, Sottas, Dujérine, and Knapp quoted by him. Lloyd<sup>2</sup> also refers to such an one in his own experience. Biernacki<sup>3</sup> reports three cases in full, two of which were syphilitic. Embolism, on the other hand, can practically be excluded, owing to the narrowness of the spinal vessels, the long and tortuous course they pursue, and the slowness of the blood-stream. Experimentally, however, by the injection of inert powders into the circulation of lower animals it has been produced by Lamy,<sup>4</sup> Sanger,<sup>5</sup> and others. Though atheroma is infrequent in the spinal circulation, syphilitic cases commonly show endo-arterial and peri-arterial changes. The same are likely to occur in infectious diseases and in many blood states, and favor thrombosis.

The softened area resulting from thrombotic occlusion of the supplying artery, just as in the brain, is likely to become hemorrhagic through the venous back-pressure, and, therefore, may present any degree of hemic discoloration. Into it a neighboring blood-vessel may rupture, with hemorrhagic results.

This condition, as Latapou<sup>6</sup> has well pointed out, comes on rather abruptly without prodromal malaise, fever, or infection. Commonly there is a tendency for the symptoms to increase often by sudden additions and extensions. At first commonly unilateral, the condition tends to become bilateral and the sensory and motor defects symmetrical. The girdle sensation and impairment of the sphincters are less marked and later developed than in acute myelitis, and bedsores less likely to appear. Leucocytosis is wanting. The treatment should be to strengthen the general circulation and use those remedies which favor resolution of local exudates. Most of these cases are syphilitic and require iodid and mercury. In other respects the management and prognosis is the same as that of myelitis.

### MYELITIS.

Under the term myelitis a host of spinal lesions have been grouped which have in common the appearance of a local softening, with more or less inflammatory disturbance. Inflammation of the cord-substance is probably never a primary process. Infection may readily reach the cord by the vascular supply. That it does so is evident in the inflammatory lesions of the cord-substance that so often follow the exanthemata and septicemic diseases generally.

The term myelitis is a generic one. In this chapter we are dealing with the indiscriminate lesions of the cord-substance, and, therefore, reserve for separate consideration the variety of myelitis known as acute

<sup>1</sup> "Relation of Diseases of the Spinal Cord to the Spinal Blood-vessels." London, 1866.

<sup>2</sup> "Nervous Diseases," by American Authors, Philadelphia, 1895.

<sup>3</sup> "Deut. Zeit. f. Nervenk.," l. c. 1894.

<sup>4</sup> "Archives de Neurologie," 1894. <sup>5</sup> "Deut. Zeit. f. Heilk.," l. c. 1897.

<sup>6</sup> "Leit. Nerv. and Med. Dis.," April, 1895.

anterior poliomyelitis that angles on the anterior gray matter, is largely confined to it, and presents a distinct clinical type. We may distinguish a *transverse* myelitis, one that is *disseminated*, and a *central* form depending merely upon the accidental location of the lesion or lesions in the cord. Of these, acute transverse myelitis may be taken as a type, and is the one commonly encountered.

**Etiology.**—Acute myelitis may follow trauma of the cord, absorption of its substance, from hemorrhage, or from fracture-dislocations of the vertebrae. It has followed violent muscular efforts, spinal concussion, and falls on the back, but in such cases minute myelitic hemorrhage or other structural lesion may have introduced the poison. It has been repeatedly attributed to cold and exposure, and this assertion has been handed down so persistently that it seems a permanent fixture in the literature. If cold plays any part, it is, as in pneumonia, to favor the introduction of infection. Compression of the cord by disease of the spine or the meninges, or by new growths, causes a localized softening which may girdle the cord. The association of myelitis and meningitis is shown in the condition of meningomyelitis already described. The extension of the inflammatory process to the cord is a clearly recognized feature of most meningeal infections. It may follow all the acute infectious diseases; probably by an initial thrombosis. Syphilis frequently leads to it, and usually by a thrombotic process. It may also result from a gummy tumor or from syphilitic meningitis. These will be considered more at length in the chapter on Syphilis of the Nervous System. Influenza, gonorrhea, all infectious and pyemic conditions, and chicken disease have caused it.

**Morbid Anatomy.**—On inspection an inflamed cord presents a red, swollen appearance and a reduced consistency that may make it pulsy and even diffident. The vertical dimensions of the softening vary from one-half an inch to several inches, and usually embrace the full thickness of the cord. Depending upon the amount of extravasated blood and the age of the lesion, the myelitic portion is red, yellowish, or white. It is usually difficult or impossible to distinguish the gray from the white portions of the cross-section, and commonly the softening is so great that the cord breaks down under the slightest handling. All details are then obliterated. *Microscopically*, there is more or less disintegration of the cord-elements. There is usually present an abundance of phagocytic elements, and amyloid bodies are frequently encountered. The axile cylinders are destroyed, or divided or granular. Sometimes a few of them appear much swollen. The nerve-cells participate in the destruction, and those that are recognizable appear swollen, pigmented, granular, filled with fat-globules, or vacuolated. Their processes early disappear. The vessels are altered, their walls thickened; the perivascular sheaths are dilated with cells, detritus, and hemorrhage. The interstitial tissue is exaggerated in proportion to the duration and intensity of the disease, and in the disseminated form of myelitis forms islets of thickened tissue. There are usually many spider-cells present. The meninges are variably affected by extension of the inflammation to them. In cases of long standing the cord may be reduced to a mere fibrous shred.



If the lesion has been of sufficient duration, ascending degenerations are found in the posterior and direct cerebellar columns, and descending degeneration in the pyramidal tracts. In addition, by the process of contiguous extension the myelitis may propagate itself in either direction from its initial focus along any of the tracts of the cord, or along its gray substance, irrespective of the direction of conduction in the physiological pathways.

In disseminated myelitis small foci of inflammation are scattered throughout considerable portions of the cord, presenting the same minute changes as outlined above. It may require the microscope to detect them, or they may be manifest as small red or hemorrhagic points in the cross-section. In the central form there is cellular infiltration about the central canal, which is often dilated and choked. The nerve-roots arising from the focus of inflammation show neuritic changes and degeneration, with corresponding muscle changes. Should a myelitis be infectious from the first, or subsequently infected by pus-producing bacteria, abscess formation results. From such a cord-abscess the mening-es may become infected and a purulent meningitis ensue.

**Symptoms.**—The symptoms of myelitis are as diverse as the cases, and each case varies with the vertical or transverse location of the lesion or lesions, with their number, extension, severity, and character. The onset is modified by the initial cause of the disease in the cord. When hemorrhage is the first step, it is apoplectic in suddenness. Traumatism has its own history. The infectious diseases have their individual clinical features upon which the myelitis is grafted. In less well-marked antecedent states the onset of the paraplegic features of the disease may be unheralded by any subjective or objective phenomena. A few days of malaise, of slight fever, or of fleeting paresthesia may indicate the systemic condition which eventuates in myelitis. In other but rare instances convulsions, high temperature, and rigors declare the toxic process and usher in the spinal symptoms. These consist usually at first of intense pains, which may be darting in character, extending along the limbs or girdling the trunk. There is more or less tingling and numbness. The distribution of such sensory disturbance in relation to the cord-segments should be significant. Shortly after—in a few minutes in hemorrhagic cases, in a few hours or a few days in infectious forms—more or less paraplegic involvement is developed, which involves everything below the segmental location of the disease. The motor loss may be sudden or gradual, complete or partial, but usually is insidious, progressive, and does not reach an absolute degree. The control of bladder and bowels is usually disturbed early, with more or less incontinence or retention. In some instances the motor features come on and progress with the sensory disturbances, or even precede them. The order of symptoms depends upon the portion of the cord first and most diseased and the destructive or irritative character of the lesions. It follows that spasmodic twitching of the limbs may occur, but, as a rule, there is complete flaccidity at first.

The number and variability of the symptoms are so great that they can best be presented under several heads.

**Sensation.**—The upper level of the sensory disturbance is usually marked by a hyper-sensitive band corresponding to the upper segmental extent of the cross-lesion, and due to its irritant action. This is always present in cross-myelitis, and should be diligently sought, as it is of the greatest localizing importance. In the entire area below the hyper-sensitive zone sensation is more or less blunted and may be completely lost in all its modes. When the cross-lesion is less complete, sensation or motion, or both, may be only partially involved. When the lesion is practically central, we have the peculiar dissociation of touch-sensations that marks lesions in this location. There is analgesia and loss of temperature sense, with preservation of tactile perceptions. Involvement of the posterior roots and extension of the inflammation to the meninges are marked by local pain and tenderness over the spine at the level of the lesion and above it. The girdle pain has the same topographical significance as the hyper-sensitive zone, and usually corresponds to it. The patient often complains of parasthetic sensations below the lesions, even in completely anesthetic territory, or in the abdominal viscera. These may be misleading to both patient and physician. The greatest care must be exercised in testing the cutaneous and other sense perceptions, as indicated in Part I. The sensory symptoms frequently vary greatly during an attack unless the lesion completely severs the cord. The reappearance of sensation where previously wanting is a good sign, just as the increase of sensory loss is indicative of extension of the disease and of bad import, sometimes of fatal significance if toward the upper cervical levels.

**Motion.**—The loss of motion corresponds to the same segmental distribution as the anesthesia, and may be complete or partial. The anterior gray matter in the inflammatory focus is usually completely destroyed, or at least its functions are completely inhibited for the time. The muscles controlled by this portion of the cord are paralyzed. The amount and degree of paralysis below the lesion depend upon the completeness of the cross-lesion, and varies within wide limits. In the disseminated and multiple forms of inflammation various functionally related groups of muscles may be singled out, as in acute poliomyelitis. Here, also, the motor loss may show any degree of incompleteness. The loss of sphincter control is usually present from the first and persistent. When the lumbar cord is affected, incontinence of urine and distention of the bladder and bowels follow the destruction of their spinal centers. The distribution of the paralysis is, therefore, always dependent upon the segment or segments of the cord that are diseased, and has a common tendency to paraplegic distribution, involving both sides more or less symmetrically. The distribution of symptoms in rare instances suggests the Brown-Sequard syndrome due to a lateral hemisectional division (see p. 36).

**Reflexes.**—The reflexes whose spinal centers are situated in the inflammatory focus are abolished. Below that level they are usually diminished at first, but at the end of a week or ten days commence to increase in vigor and gradually attain extreme exaggeration. Should the lesion actually divide the cord, they are abolished, as in traumatic cases, but the preservation of a very few fibers in any portion of the



cord's cross-section seems sufficient to allow of their exaltation and the development of spasticity and rigidity. All forms of clonus and intensification of reflex activity may be encountered. Babinski's toe-sign is commonly present. The tendency is for the lower limbs to be rigidly extended, adducted, and sometimes crossed. Less frequently flexion predominates and the legs are flexed upon the thighs, which are firmly applied along the anterior surface of the trunk. The clonic condition of the limbs sometimes serves to jerk them about sharply upon slight skin friction, even by the removal of the bedding or any gentle manipulation. The flaccidity of the early days is replaced by a hypertonicity, so that the muscle-masses may stand out prominently. Contractures may result and often do. Priapism is commonly present in cervical myelitis, and frequent inflammation of the dorsal cord, but absent in lumbar involvement. Lesions in the cervical region are commonly attended by a dilated pupil, but in some cases, especially of the disseminated variety, optic neuritis is present, and pupillary responses are variously modified. The condition of the vesical and rectal reflexes is one that should early engage attention. If the lesion involves the lumbar cord, the sphincters are usually relaxed and incontinence follows, but there is a tendency to urinary retention through relaxation of the visceral walls, and cystitis is easily established. Again, when the lesion is above the lumbar cord the sphincters operate automatically, and both feces and urine are discharged at intervals; but, again, the bladder-wall is likely to yield, or acts ineffectually without the aid of the abdominal muscles; urine is retained, becomes ammoniacal, and, through contamination by catheter or otherwise, cystitis develops. Rupture of the bladder through distention and ulceration, causing peritonitis, has been seen. Some cases present a most obstinate and extensive gaseous intestinal distention. This occurs most frequently when the lesion is above the mid-dorsal region.

**Trophic Changes.**—The muscles that are anatomically related to the diseased cord-segment waste promptly and show the reaction of degeneration. In addition the inert limbs lose in size from disuse and considerable emaciation is frequently presented, but the electrical changes are lacking and the reflexes are usually increased. The general vasomotor and trophic conditions below the lesion are disturbed. A slight stroke of the finger-nail upon the skin will usually present a line of persisting vascular stasis like the meningal taché, and at first the paralyzed portions show an elevation of temperature even above that in the mouth. In cases of long standing the local temperature is raised and the paralytic members are blue, cold, cyanotic, and often edematous. The skin is often dry, hard, and scaly, and readily breaks down under pressure, forming ugly, sluggish, unmanageable bedsores. The tendency to bedsores is prominent from the first, and it is in these cases that the serum is sometimes denuded within a few days under the continued influence of pressure, irritation from urine or feces, and the dystrophic element. The entire vesical mucous lining may exfoliate from disturbed trophic conditions. Joint-lesions of the arthropathic sort are rarely induced.

The general nutrition of the patient suffers to some degree, but less than would be expected, and it can usually be maintained at a reason-

side level. The danger arises from extension of the myelitis and from complications arising through cystitis, bedsores, nephritis, and septicæmia, or concurrent acute infections, such as pneumonia.

**Course.**—Acute cases reach their maximum in a few days, others in a few weeks, and then, if death does not result, a long stationary period or one of gradual improvement or decline succeeds. The occurrence of an extension of the inflammation may, at any time, jeopardize life by invading the respiratory apparatus. Acute bedsores is always a dangerous complication, and cystitis is hardly less so. When spastic features develop, they rarely recede to any considerable extent, and imply permanent disability and the paralytic state. Sensation or motion may return singly. The localized wasting due to involvement of the anterior gray never repairs, and adjacent portions of the gray matter may subsequently be involved. Death may take place early from cardiac and



FIG. 331.—Atrophic bedsores with hemorrhages and necrosis in a case of transverse myelitis.

respiratory failure or follow at any period from exhaustion due to the primary infection or that secondary to bedsores, cystitis, nephritis, septicæmia, or is caused by a gradual extinction of the vital energy. The cases that recover bear the indelible marks of the disease in weakened and spastic legs, areas of anesthesia, sphincteric paresis, and local atrophies variously distributed in accordance with the seat, extent, and intensity of the cord-injury. These furnish cases of so-called chronic myelitis, but inflammation has subsided and the conditions presented are due to the degenerations that follow the primary lesion. They are more fully described under the head of The Paralytic State.

Diagnosis in myelitis presents numerous problems and requires painstaking examination and study. We have to ask ourselves: (1) Whether the cord is actually diseased; (2) the extent of that disease—namely, its localization—and (3) its origin. Unless there is loss of



certain groups of cord-functions anatomically related, we can not incriminate the cord. Of these, the most important are early loss of motion of a paralytic distribution and relaxation of the sphincters. Corresponding sensory disturbance or anesthesia surmounted by a band of hyperesthesia will almost surely be added to, if it does not precede, the motor loss. The onset is usually acute, and in the hemorrhagic variety it is sudden. Extreme pain of a radiating segmental variety and sudden onset suggest an initial vascular lesion. After ten days we may detect muscular atrophy corresponding to the lesion and usually increased reflexes below the lesion, which enter in the spastic stage that is to persist as a paralytic state if the patient survives. Even before this time the loss of faradic response in the muscles innervated by the spinal grey enclosed in the inflammatory focus may indicate the myelitis and its vertical extent. To the further localization of the cord disease we bring to bear the considerations set forth in the preceding chapter. Our main early guide is loss of reflexes and the vertical extent of such hiatus in the chain of spinal reflexes.

In *poliomyelitis* sensory deficiency is not present and the motor loss is usually of monoplegic outline. In *meningitis* we have the early and persistent root pain, tenderness over the spine, and retention of motion and reflexes. In *Lowe's palsy*, or *acute ascending myelitis*, we meet the steady advance of the paraplegia from the feet and legs upward, with undisturbed sensation, faradic activity, sphincteric control, and tendon reflexes. In *multiple sclerosis* the slow onset, involvement of all four extremities, major affection of the extensors, and parasthesia at the distal ends of the limbs are significant. In *hysteria* the sensory disturbance has a characteristic outline, the reflexes are not greatly disturbed, trophic changes are not present, and hysterical stigmata are obtainable. The mistake usually made is to overlook an organic disease because hysteria is also present.

The diagnosis of myelitis having been reached, it is always in order to question its origin. This may be evident from the history or presence of transients, acute infectious disease, septicemia, syphilis, or other infective state.

Prognosis in such a generic condition as myelitis must be based upon general rules applied to the individual case. The outlook is always grave as to life and positively bad as to complete recovery. If the patient is not carried off during the first few days by the implication of the cardiorespiratory apparatus, or by the overwhelming systemic effect of the infection, which is perhaps only locally manifest in the cord, and if he reaches the end of the first week without any indications of extension of the myelitis, he may be considered out of immediate danger. If at the end of three or four weeks he does not show at least some slight return of motion and sensation, it is not likely that these will ever greatly improve. On the other hand, distinct improvement within the first two or three weeks is usually followed by rapidly progressive gain almost to the point of entire recovery. When spasticity appears it implies descending degeneration in the pyramidal tracts and lasting disability. Cervical myelitis is almost invariably fatal. Involvement

of the dorsal cord is much less disastrous than when the lesion invades the lumbar enlargement and perfect permanently cripples the sphincter control and the legs. Acute bedsores and acute cystitis are ominous complications. Local muscular atrophies are subject to this rule: If faradic response can not be obtained in such muscles at the end of the first two weeks, they may be considered as permanently impaired. In syphilitic cases that reach a marked degree of paralysis we may hope to prevent extension of the disease and sometimes to secure a marked recession of the paresis but never for complete cure.

Treatment divides itself into three parts: (1) That of the causal condition; (2) that of the invasion stage, and (3) that of the paralytic state. Traumatic conditions, pressure from meningeal hemorrhage or new growths require surgical measures. Any systemic infection or infection atrium must be directly dealt with. For the local condition the treatment outlined for meningitis is available. During the invasion period the inflamed cord should be kept elevated by placing the patient in the prone posture. Local applications of ice are useful. Active cathartics should be used if the patient's strength warrant their administration. Quiet, and above all cleanliness, must be secured and local pressure avoided. Early attention to the bladder is usually needed but catheterization is only indicated when vesical retention can be determined by palpation and percussion, and then must be carried out with the most scrupulous antiseptic precautions. Feculent disturbance is usually a part of the original causal process and to be met accordingly.

After the first two or three days, if the case runs on, a water or air bed will be found of the greatest service in distributing pressure. The heels, elbows, and other bony prominences in the paralytic field should be carefully padded with cotton. The constant use of a bed-urinal will often help to keep the patient dry. Alimention and supporting treatment will require thoughtful attention. At the end of a week careful massage and faradization may be employed, if not contraindicated by surgical conditions in the spine. Gentle friction and kneadings of the muscles are indeed advisable from the first day, and the position of the paralyzed limbs should be changed hourly if only slightly, as is automatically done in healthy sleep. When a bed-sore develops, its best management depends upon keeping it perfectly dry. To this end a soft gauze pad and an abundance of pulverized boric acid should be used, but the dressing must not be cumbersome or so arranged as to exercise pressure and the patient's position must also conform to this requirement. Cystitis requires careful, thorough, skilful catheterization and washing out of the bladder every eight or twelve hours. The administration of salol, beta-naphthol, or urotrypin also tends to render the urine unirritating and to some extent disinfect the bowel contents. The management of the case now resolves itself into one of good nursing and attention to obviate complications, especially malposition of joints and contracture deformities.

As motion and power reappear the patient should be encouraged to use the limbs intelligently. By concentrating his attention upon a certain movement he can often produce it after a number of attempts.



Local nutrition must be kept up by massage and electricity. The use of the faradic brush to the anesthetic area is of service in restoring sensation. The general physical condition and usual constipation require constant attention.

#### THE PARAPLEGIC STATE.

The paraplegic state is the usual termination of a myelitis or any indiscriminate cord lesion that does not end fatally, and corresponds in some sense to the "hemiplegic state" that supervenes upon brain-lesions. Very often arising from such cause as acute myelitis, it is mistaken for a chronic inflammatory condition and denominated chronic myelitis or confounded with ataxic paraplegia and even with locomotor ataxia. The descending degeneration in the pyramidal tracts is a consequence of any lesion which involves the upper motor neuron. When due to bilateral lesions above the cord it is best called a diplegia. Cross-lesions of the cervical cord are usually promptly fatal, so that the paraplegic state arising from indiscriminate lesions only reaches its later and characteristic development in cord lesions below that level.

**Etiology.**—All the indiscriminate cord lesions, such as traumatism, hemorrhage, thrombotic softening, myelitis, tumors, and pressure from meningeal and spinal diseases and growths, give rise to a paraplegia which is more or less pronounced in proportion to the extent of the lesion. In addition, the so-called system lesions which are marked by selective degeneration of the lateral tracts produce paraplegic conditions, but without distinct anesthesia. We find it also in Little's disease, ataxic paraplegia, family cerebellar ataxia, multiple sclerosis, family spastic paraplegia, and amyotrophic lateral sclerosis. The only common fact is the degeneration of the motor tracts in the lateral columns. Diseases of the upper neuron arising from intracranial lesions have been considered in connection with diseases of the brain (Part III).

**Symptoms.**—The symptoms of this secondary state vary greatly and are dependent upon the nature, extent, and activity of the initial lesion. In the system diseases the spastic paraplegia, as in Little's disease, may be really a diplegia from embryonic defect, and congenitally present. In the family ataxias the defect is also hereditary, but the symptomatic development is postnatal and usually insidious. Ataxic paraplegia and amyotrophic lateral sclerosis also develop very gradually and usually in adult life. They all in common possess defects of the pyramidal tracts marked by loss of muscular control and strength especially developed in the lower extremities, and by increased reflexes and spasticity. The special paraplegic features which they possess will be taken up in the description of the various system diseases. In this present connection the paraplegic state arising from indiscriminate lesions is principally in view.

If the initial cord lesions be acute, as in traumatism, hemorrhage, and myelitis, the motor loss is promptly established and the muscles are flaccid and inert. The sphincters are usually relaxed. The reflexes represented in the diseased cord segments are abolished and those below the injured focus are diminished. If the cord is entirely severed, they are completely and permanently lost and rigidity does not ensue. When the cord is not entirely divided, at the end of a week in some cases, in

others several weeks later, the increasing reflexes indicate degenerating lateral motor tracts and usher in the spastic features that are to permanently remain.

When the inciting cause of the paraplegia is of gradual development, as in pressure conditions arising from Pott's disease and spinal neoplasms or new growths in the membranes or cord, the clinical program is very different. Root pains, girdling the trunk or stroking into the limbs, depending on the segment location of the lesion, are early symptoms and usually there is complaint of heaviness, weakness, and clumsiness in the legs. This increases either steadily or by stages, in the intervals of which some amelioration may occur, and eventually the hypersensitive girdle and subjacent anesthesia are more or less well developed. Sphincteric loss is a late feature and is usually preceded by months of slighter degrees of incontinence. As the pressure increases, compression of the pyramidal tracts gradually develops; they degenerate and the reflex activity is correspondingly exalted. It is in these cases of slow cord compression that the spasticity, reflex animation,



Fig. 122.—Paraplegia (gait).



Figs. 123 and 124.—Gait in spastic paraplegia due to syphilitic myelitis, showing rigidities, forced knees, and adducted thighs.

and muscular rigidity with resultant contractures reach their highest exemplification.

When the acute cases have improved enough to walk or the indolent cases have attained a fair degree of development, the gait is highly characteristic. The patient's feet once planted seem glued to the floor, and the upper portion of the body is inclined forward in advancing. The pelvis is elevated on one side and that limb is then brought at



dragged to a position under the center of gravity like a rigid pendulum (Fig. 132). It may even be aided by the hand. As it advances it is shaken by clonic movements, and when planted these may cause it to execute several dancing steps before it is securely placed, during which the heel is forcibly elevated and the patient may be bodily jostled up and down. The body is then again inclined forward over the supporting limb, and the opposite member is in turn carried a little in advance of its fellow. These short, jerky, halting, clonically disturbed steps are frequently rendered more difficult by the overaction of the adductors of the thighs which displace the limbs inward, cause the knees to interfere with each other, and sometimes even induce a cross-legged gait. Progress, in spite of the disturbance of the gait, is usually made in a fairly straight line, unless some slight obstacle over which the patient is sure to stumble, or an unusual amount of clonus, cause him to swerve. In nearly every respect the gait is the opposite of the stepping of multiple neuritis.

If walking is impossible, the attitude in standing may still indicate



FIG. 133.—Paraplegic from spinal lesions. Attitude in bed. Thighs abducted and crossed. Feet locked out.

the spastic state. The tendency to contracture usually draws the knees forcibly together and partially flexes the knee- and hip-joints (Figs. 133 and 134). A sudden reflex contraction of the calf-muscles may cause the patient to rise on his toes or throw him to the ground. The knees often suddenly give way.

When the patient is bedridden and the paraplegic state is developed to an extreme degree, the lower limbs may be rigidly extended on the pelvis and at all their joints. Adduction is strongly marked and may even cross the limbs. If one foot be lifted from the bed, the rigidity may serve to cause the other one to follow, as if the hip- and knee-joints were ankylosed. In other severe cases flexion predominates, and the lower extremities are rigidly folded upon themselves and upon the trunk, so that the heels are drawn up to the buttocks, the knees to the sternum.

The reflexes are always exaggerated after the early flaccidity of acute cases has receded, and in the later stages become excessive to an incredible degree. The slightest tap on the patellar tendon forcibly throws out the leg, and the whole extremity may be seized with a clonic activity that provokes jerky, more or less rhythmical, movements which may also appear in the opposite limb, and finally end by a jack-knife contraction that violently flexes both extremities upon the trunk and at the knees. These reflex storms may even follow the slightest cutaneous impression, such as gently removing the bed-clothing, and are not infre-

quently the source of much pain and suffering. The toe-sign is prominent. The vesical and rectal reflexes usually work automatically in protracted cases of paraplegia, and are only slightly under the patient's control, or entirely beyond it. Lesions in the lumbar cord may destroy them. When the upper dorsal or lower cervical cord is affected, priapism is frequently present, and cervical lesions cause dilatation of the pupils.

All of these conditions vary from the slightest to the most extensive degree in different cases. Slow compression of the cord, as by tumor, gives the most highly colored picture, ataxic paraplegia perhaps the least.

The nature and character of the sensory disturbance naturally depend on the extent of damage to the posterior half of the cord. Atrophy of muscles is in the same way dependent upon the destruction of the anterior gray and is related to the vertical extent of that destruction. More or less general emaciation, due to inactivity and depressed systemic conditions, is commonly present. Contractures produce deformities that still further cripple the patient.

The prognosis naturally depends on the character of the cord lesion. The removal of pressure in Pott's disease and in spinal tumors by operation, frequently results satisfactorily and the cord functions return to a normal state. When the spasticity is the result of destructive lesions of the cord, as in myelitis and trauma, some disability is bound to persist. It may eventually be very slight or it may progressively increase. Every case must be individually considered.

**Treatment.**—The treatment of the paraplegic state consists of: (1) Removing the cause if possible; (2) preventing contracture distortions or converting them by surgical means, such as tenotomies and orthopedic apparatus; (3) the use of electricity and massage to atrophied muscles; (4) general measures to build up the systemic condition; (5) local applications over the spine to control any lingering inflammatory state or to hasten absorption of exudate, and (6) of the administration of spinal sedatives to reduce the reflex excess. Local conditions, such as indolent ulcers, cystitis, and incontinence, furnish their own indications. It will often be found that massage and electricity produce so much reflex stimulation that they must be discontinued. Hot baths often produce a grateful relaxation. Counterirritation to the spine and the resources of hydrotherapy sometimes are of great advantage. Following Forrester's<sup>1</sup> plan many cases have been treated with great benefit by the division of the posterior nerve-roots.

#### LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS).

The case observed by Landry,<sup>2</sup> reported in 1859, presented those striking peculiarities: An acute paralysis beginning in the legs, extending to the trunk and arms, soon involving the bulbar centers, and terminating fatally. The paralysis was not marked by loss of sensation; the sphincters were not involved. The muscles retained their faradic irritability, the mind was not disturbed, and the temperature was practically normal. No changes were found in the central nervous apparatus by microscopical examination, but the spleen was acutely enlarged.

Since that time several hundred cases have been reported as instances

<sup>1</sup> *Zeitschr. f. Orthoped. Chir.*, 1903.

<sup>2</sup> *Gazette Heb.*, 1859.



of Landry's paralysis, some of which correspond closely to, while others depart materially from the original outline. Of these latter we may say that most of them were cases of multiple neuritis, some were cases of poliomyelitis anterior, and others were cases of cross or disseminate myelitis with extension. With improved methods of investigation there has gradually accumulated a considerable number of cases which are clinically true to the prototype, but show organic disease in the spinal, lular, and even in the cerebral matter and in the nerve-roots and peripheral nerves. With the development of bacteriology we may now add typical cases in which infectious bacteria have been observed in the spinal cord or obtained by cultures from it. We are, therefore, justified in defining Landry's paralysis as an acute infectious ascending paralysis due to an infectious or toxic condition that may induce a myelitis largely confined to the anterior gray matter of the cord and which may, in addition, cause root involvement, peripheral neuritis, and changes in the medulla and cortex of a similar nature to those in the cord. It is not unlikely that the entire symptom-group of Landry may eventually be classed as acute poliomyelitis with ascending features. Such cases occur in every epidemic outbreak of that disease.

**Etiology.**—This form of spinal palsy is about four times as frequent in adults as in children, according to the tabulation of selected cases by Bailey and Ewing,<sup>1</sup> and affects males nearly three times as often as females. It has followed close upon or occurred during attacks of numerous infectious diseases and conditions. Small-pox, tuberculosis, typhoid, pneumonia, diphtheria, syphilis, influenza, pelvic cellulitis, the puerperium and obscure febrile disturbances have seemed to play a part in its inception. Alcoholism<sup>2</sup> and exposure to cold are also rather frequently mentioned, but in numerous cases not the slightest cause has been detected and the patient apparently was in good health previous to the paralysis. The toxic features of many of the alleged causes jump with the clinical manifestations which are highly significant of an infectious state, and coincide with the few bacteriological findings that have been reported. The idea of a toxin which has an elective action for the spinal gray producing first dynamic conditions, later followed by histological changes, best explains the various post-mortem findings and the clinical manifestations of the disease. E. F. Buzzard<sup>3</sup> isolated a micrococcus in pure culture from the blood of a case of Landry's paralysis and apparently the same organism was found in large numbers in the dura mater of the same patient. Subdural injections of the cultivated organism produced rapidly spreading paralysis in a rabbit and the organism was again obtained from the blood and dura. The changes in the nervous system of the patient and rabbit were of the kind produced by toxins and in neither could the microbe be demonstrated in the nervous structures or even in the pia arachnoid. That the infection or toxin reaches the cord through the blood is abundantly shown by the perivascular changes, and the frequent limitations of the myelitic invasion to the territory irrigated by the anterior median arteries and the vessels reaching the anterior horns along the motor roots.

<sup>1</sup> *N. Y. Med. Jour.*, July, 1890.

<sup>2</sup> Kerner, *Zentralbl. klin. Med.*, B4, xxix.

<sup>3</sup> *Trans.*, Spring, 1901.

**Morbid Anatomy.**—In the older reports, as in the first instance, no morbid changes were detected anywhere in the nervous system. As methods improved and data multiplied, reports of myelitic softening, especially in the gray matter, but also implicating the neighboring white fibers of the dorsal and cervical cord, degeneration in root-fibers and peripheral nerves, and, finally, changes in the medulla, cerebellum, and cerebellum were made. A majority of cases, whether showing cord changes or not, presented an acutely enlarged and softened spleen and often engorged lymphatic glands. Of late no case that has been systematically and competently examined has given negative findings. The cellular structures of the anterior gray or the cylinder processes arising in the motor cornal cells are found disturbed. Eisenlohr, Ross, Hoffman, Immersman, Curschmann, Koth, Hlaven, Marinresco, Bailey and Ewing all found such changes varying from fragmentation of the cylinder process or slight swelling of the cell bodies and chromophilic changes of the cell protoplasm to well-defined poliomyelitis and diffuse cellular infiltration throughout the spinal gray. Frequently the blood-vessels show a perivascular small-cell infiltration and the motor cells present marked degenerative changes. In cases of sufficient intensity and duration the peripheral nerves are degenerated and muscular degeneration and even atrophy are added. The cranial nerve nuclei and the cellular and vascular elements of the cerebral and cerebellar cortex were similarly affected in Ewing's case.<sup>1</sup>

Bacteriological examinations have sometimes been made with negative results and suitable stains have often failed to show bacteria in the cord sections. Reilinger<sup>2</sup> met with this experience even when cultures from the cord readily developed streptococcus pyogenes and streptococci were also found in the lymph-spaces of the gray matter of certain other portions of the cord. Eisenlohr found the staphylococcus pyogenes aureus in all cultures and staphylococcus aureus in cultures from the spleen. Cutroni found a rounded bacillus in the peripheral nerves. Guzzetti found chromogenic bacilli in cord cultures. Marinresco found cocci in the ganglion-cells. Roger and Josic<sup>3</sup> have demonstrated the pneumococcus. Thoinet and Masseline have produced spinal paralysis in rabbits by the intravenous injection of staphylococcus pyogenes aureus and of bacillus coli. With the newer methods and findings in poliomyelitis to guide, it is probable that the identity of Landry's paralysis with the infantile variety will be proved in many cases.

**Symptoms.**—Acute ascending paralysis may develop during an attack of some infectious disease or may follow it. In several epidemics of poliomyelitis such cases have been encountered. Frequently, however, it comes on without malaise, fever, or preliminary symptoms, usually without tingling, numbness, or other sensory disturbance. A feeling of weakness begins in the feet and legs, and slowly creeps upward, becoming more and more pronounced in the lower levels as the disease mounts. It may affect one leg first or most. At the end of two or three days or a week the lower extremities are completely paralyzed and the weakness has involved the trunk and upper limbs. The

<sup>1</sup> *Loc. cit.*

<sup>2</sup> *Congress. Rendus de la Soc. de Biol.*,<sup>3</sup> April 1906.

<sup>3</sup> *Presse Méd.*,<sup>1</sup> July 27, 1906.



breathing becomes superficial from involvement of the thorax, and difficulty in swallowing soon appears. In severe cases every voluntary muscle below the face is completely paralyzed and relaxed, and even the cranial nerves may be involved, especially the oculomotor, facial, and hypoglossal. Cerebral and mental symptoms are absent until the dyspnea or cardiac failure is pronounced and induces them. The sphincters are not, as a rule, relaxed; there is no tendency to bedsores or dystrophy; the tendon and superficial reflexes are usually present; the electrical responses are normal; and sensation, together with the special senses, is not perverted. If a fatal issue do not occur, the symptoms of paralysis slowly recede in the reverse order of their appearance, and when they have distinctly subsided from the upper levels recovery may be anticipated.

In some cases the onset is reversed, the upper extremities first showing weakness; and, indeed, the ordinary type may be greatly modified, as can be readily understood from the varying anatomical distribution of the organic lesions in well-authenticated observations. In two cases falling under the writer's attention, the clinical history was typical, complete wasting of isolated muscle-groups in all four extremities occurred, and persisted for years, without any appearance of ultimate improvement. Paresthesia and dysesthesia are not rare, and anesthesia may gradually follow the paralytic invasion, advancing in a similar manner. The reflexes may also subside and disappear in an ascending progression. Even electrical modifications and the reaction of degeneration are encountered. The progress of the paralysis may stop at any point, and then recede. A temperature of  $101^{\circ}$  to  $103^{\circ}$  F. has been observed, but, as a rule, it does not rise above the normal. Profuse perspiration sometimes and splenic enlargement frequently are encountered and bespeak the toxic state.

**Course.**—The course from inception to fatal termination may be very brief,—less than two days,—and fatal cases usually end within ten days. Prolonged cases may only reach their acme in a month. After a stationary period of varying length in the hopeful cases, improvement takes place usually in a retreating order, but convalescence is slow and may require months. On the other hand, it may be rapid, or, as in the cases previously mentioned, permanent injury may result.

**Diagnosis.**—The diagnosis in some cases must necessarily be extremely difficult, but in the typical form is readily made, providing the existence of this rare disease is kept in mind. It rests upon the method of invasion, the pure motor paralysis, the comparatively negative conditions as to reflexes, sensation, and electrical reactions, and the history of some possible toxic state. Some cases are complicated by bacteria, which is capable of greatly obscuring the diagnosis. When slight electrical changes and paresthesia are present, it is impossible to exclude neuritis, and the occurrence of peripheral nerve-lesions in some instances has already been pointed out. In meningitis the pain and rigidity are distinctive. In cross-myelitis we have all spinal cord functions involved below a definite level and lack the ascending features.

**Prognosis** is always grave, since even in the irregular and prolonged cases one can not foretell at what moment further symptoms may appear,

and the main danger to life depends on their presence. Rapidly ascending symptoms imply a speedy termination, but there is no invariable rule. Only when the tide has turned and symptoms are receding can one entertain a reasonably hopeful prognosis. The presence of neuritic conditions or of electrical changes implies a prolonged convalescence and doubt as to ultimate complete recovery. Where cerebral symptoms appear they are of bad import, signifying either profound toxic conditions or the near approach of death from cardiac or respiratory failure.

Treatment will be directed against any general toxic condition present or reasonably suspected. The salicylates, tincture of the chlorid of iron in full doses, bichlorid of mercury to the point of toleration, thorough cleansing and disinfection of the alimentary tract, supportive diet, conservation of nervous energy and strength, are valuable. Urotropin in 10-grain doses every six hours may be expected to have a beneficial influence upon the cord infection. Spinal puncture is always in order both for diagnostic and therapeutic reasons. To the spine a *raffin* sinapism the whole length of the back, frequently repeated, is of service; even the thermocautery is advised by some. The paralyzed limbs should be gently massaged to improve circulation and give comfort. When swallowing becomes difficult or impossible, feeding by the stomach, nasal, or rectal tube must be adopted, and the preference is for the nasal tube, providing care be exercised to avoid passing it into the larynx. During convalescence, massage, electricity, local douches, tonics, generous diet, and general measures are the main reliance.

#### CASSON DISEASE, OR DIVERS' PALSY.

Workmen and others subjected to high atmospheric pressure, as in descending to great depths in diving apparatus, or in making certain excavations by caisson construction, are frequently affected with cerebral symptoms and paralytic conditions of mainly a paralytic character. The disturbance varies in intensity from slight giddiness and neuralgic pains to paraplegia. Even sudden death may occur. The symptoms appear while the air-pressure is being reduced, or within the following half-hour. In a minor degree, high altitudes, as in mountain climbing and ballooning, furnish analogous conditions and symptoms.

**Etiology.**—The cause of divers' palsy is not so much the increased atmospheric pressure, as its sudden reduction. A number of predisposing causes have also been fairly determined. Advanced age, alcoholism, heart and kidney disease, obesity, hunger, and any condition of physical depression furnish a liability to its onset. On the other hand, those who have for months been gradually subjected to increasing air-pressure acquire in some degree an immunity by habituation. The length of exposure and the amount of pressure are followed by proportionate effects, but symptoms rarely result unless the pressure reach two atmospheres—thirty pounds.

The mechanism producing the palsy or the slighter symptoms is furnished by the vascular apparatus. Various theories have been advanced to explain the results. One supposed that during the time of increased air-pressure the superficial and peripheral parts of the body are engorged and the central organs actively congested to a similar degree,



and this congestion reached such a point that the capillary field became paretic from distention, and could not promptly deliver itself of its superabundant blood when pressure was removed from the periphery. A passive congestion then was supposed to ensue, with a stagnant blood-current. The lower cord is placed at an especial disadvantage through its arterial arrangement, which mechanically conduces to maintain the vascular stagnation. Serous effusion into the cord and meninges thus followed, or the effusion might be hemorrhagic.<sup>1</sup> This idea of increased arterial tension may be entirely discarded, as careful tonometric examinations before, during, and after working in compressed air clearly disprove it.<sup>2</sup>

A more important feature is the evolution of the intra-vascular gases. The oxygen, nitrogen, and carbon dioxide of the blood, compressed under the high pressure, are liberated by restored low pressure, and, expanding, fill the vessels with gaseous bubbles, producing something like an air embolism. The gases also escape into the tissues. Celsus, of Athens, has seen gas bubbles in the vessels and even in the cord parenchyma of dogs subjected to high air-pressures, and gas has been seen in the tissues and vessels in some human autopsies after death from this cause. M. A. Starr, in his book on Nervous Diseases, photographically illustrates this condition in both the brain and spinal cord. If vascular accidents do not follow, the circulation is gradually equalized, the gas is absorbed or removed by respiration, and symptoms subside. P. Bert, Huché,<sup>3</sup> Snell,<sup>4</sup> and Basso<sup>5</sup> have practically proved the gas theory. Edema, effusions, and hemorrhages naturally produce more or less lasting symptoms in the brain, and especially in the cord.

**Morbid Anatomy.**—In the few autopsies on record, which have all been made some days or weeks after the onset of the attack, the cord has always been found abnormally congested. Small hemorrhages have been seen and diffuse myelitis with degenerative tracts have been noted. In some cases there has been an edematous condition of the membranes and cord. Hemorrhages into parenchymatous organs and mucous surfaces have also been observed. The secondary myelitis is most pronounced in the dorsal half of the lumbar cord and the anterior horns are practically unaffected. In other words, that portion of the cord which has the best vascular supply, and where stasis and edema would first subside, ordinarily escape injury. The painful manifestations of the attack are perhaps explained by the location of the vascular disturbance in the sensory portion of the cord.

**Symptoms** arise as the air-pressure is being reduced, or shortly thereafter, and may appear while the men are in the locks or chambers that are placed between the various pressures or after the patient has gone some distance in the open air. At first the cerebral features predominate; headache, giddiness, faintness, nausea, vomiting, delirium, double vision, and even coma may precede or accompany the spinal

<sup>1</sup> Hirt, "Handbuch der spec. Psychologie und Therapie," vol. i.

<sup>2</sup> H. Brooks, "Med. Rec.," May 25, 1907.

<sup>3</sup> Berlin klin. Wochens., May 31, 1907.

<sup>4</sup> "Compressed Air Illness," London, 1906.

<sup>5</sup> "Compressed Air Illness," Report of Commission on Occupational Disease, Chicago,

symptoms. These consist of paroxysmal pains, frequently of great intensity. These pains are known as "bends" among the workmen. They usually are felt in the legs, but may affect the trunk or upper limbs. Their relation to the posterior portion of the cord has already been mentioned. Soon the patient feels numbness and weakness in his legs, which may increase rapidly to complete paraplegia, usually confined to the lower extremities, but it may extend downward from any spinal level. It involves motion, sensation, and the sphincters, and presents clinically the features of a cross-section of the cord of rapid onset. In extreme cases the patient falls dead, or, after staggering a little, falls and expires with or without delirium and convulsions. Symptoms may be most variously combined and show any grade of intensity. The majority recover promptly. Of those coming under treatment about one-half recover, about half have lasting paralysis, and about three per cent. die. Those who do not recover within the first three or four days present the symptoms and run the course of a lumbal or dorsal myelitis of varying extent and severity. In a study of 316 cases occurring during a period of several years in New York State<sup>1</sup> lists the frequency of symptoms as follows: Bends or myalgia, 106; aural symptoms, often with vertigo and rupture of drum-heads, 65; pain in the joints, often with swelling, but no inflammation, 40; acute paraplegia, 26; monoplegia, 17; cerebellar symptoms, 14; apoplexy or syncope, 13; aphasia, a few.

**Treatment.—Prephyphaxia.**—Persons who are to be subjected to increased air-pressure should be rigidly examined. Those presenting the predisposing conditions mentioned should be excluded. Only hardy young men with sound hearts and regular habits should be accepted for this work, and spare individuals are preferable. In case of an extensive undertaking the same men should be employed throughout the task, for they may be habituated to the increasing pressure as the work progresses, thereby gaining immunity. In a pressure of over thirty pounds they should work short shifts of two hours or less, and plenty of time should be taken in passing the locks. Smith<sup>2</sup> says five minutes for each fifteen pounds of extra pressure. Inexperienced persons should take much more. It is well not to enter the pressure-faring. Small lays stress on ventilating the works to reduce the amount of gases in the circulation as well as on general principles.

**Treatment of the Attack.**—If symptoms arise, the patient should be hurried back into the cabin, and if the symptoms subside, as they often do, very slowly returned to the outer air. Morphin is often required for the intense pain. Ergot in drum doses of the fluid extract every hour will sometimes relieve the pain and apparently check the disease. It has been suggested to bandage the limbs, and even the trunk, thereby restoring something of the surface pressure and maintaining the spinal circulation. When paralytic features have developed, the treatment is that of myelitis.

#### TUMORS OF THE SPINAL CORD AND ITS VARIOUS ENVELOPES.

New growths arising in the spinal meninges or on the inner surface of the bony canal or in the cord itself produce definite symptoms only as the

<sup>1</sup> "Med. Rec." June 19, 1909.

<sup>2</sup> Pepper's "System of Medicine," vol. ii.



cord or the nerve-roots are disturbed. They may be properly grouped together because of their common symptomatology, which renders a positive clinical diagnosis as to their original sites impossible, and makes every operation for their removal in a limited sense an exploratory one. The importance of an early diagnosis of these tumors is emphasized by the fact that most of them can be successfully removed if taken in time, and the fatality, which otherwise attends them, averted. The paraplegia to which they give rise may in the same way be prevented, and in some instances caused to recede when not too far advanced.

Tumors arising from the meninges and extracordal structures are about six times as numerous as those primarily cordal. Sarcoma and its varieties furnish more than one-third of the tumors, tubercle and echinococcus each about a tenth, and carcinoma and all varieties of benign growths, including gumma, the balance. Glioma, which is so common in the brain, also appears in the cord. It is usually, however, distributed closely about the central canal or in longitudinal bands in the substance of the cord, where, undergoing degeneration, it produces channels or false canals. These give rise to definite symptoms and a clinical type, which is called *myelomyeloma*. It is described in a later chapter.

Tumors affecting the spinal cord are usually of small dimensions, owing to the rapidity with which they destroy the cord and lead to a fatal issue. They are commonly single, but several tubercular growths and multiple sarcomata and neuromata have been reported. The favorite location of spinal growths is in the dorsal and lower cervical regions. Regarding the causation of spinal tumors, the same ideas exist as pertain to the development of tumors elsewhere. Traumatism is often alleged and may undoubtedly serve to locate a syphilitic process and perhaps to favor the invasion of tubercle and angiomatous growths. Its relation to malignant neoplasms is largely supposition.

**Morbid Anatomy.**—The post-mortem examination reveals the new growths arising from the extracordal tissues, the membranes, or the cord, in the substance of which it may rarely be embedded.

It frequently is traversed by several nerve-roots, or these may be destroyed. It may have a vertical extent of several inches. From pressure upon the cord there is a zone of softened cord-substance which frequently shows an inflammatory condition. The cord may be much indented by the growth, or compressed almost to complete division. Changes in the cord substance due to pressure-atrophy, softening, and myelitis are present, with resulting secondary



Fig. 100.—Tumor of the cord (epidural).

degenerations related to the portion of the cord that is affected. The consequences of myelic softening and myelitis are found in the muscles, bladder, kidneys, etc., depending in distribution upon the anatomical and clinical features of the given case. The various growths are marked by their ordinary individual characteristics and histological peculiarities. Subdural cysts containing clear fluid are sometimes encountered either alone or in association with tumor formations. When existing alone, Oppenheim denominates the condition *meningitis serosa spinalis circumscripta*, and several such cysts may be found in a given case, in some instances blocking the flow of spinal fluid, especially downward, and even causing pressure changes in the cord. Tumors and cysts, especially when located in the cervical region, may be attended by more or less hydrocephalus, with its complicating symptoms.

**Symptoms.**—The initial and most common symptom of spinal tumor is usually *pain*. This is of two varieties: first, that resembling irritation of the posterior roots, and, second, that due to disturbance of the sensory tracts in the cord. The first gives rise to pinching sensations and partially follows in distribution the fields of the peripheral nerves. The second follows the segmental outlines and may also give rise to pains referred to parts below the lesion whose sensory pathways in the cord are irritated. If these pathways are broken, the pain may be referred to an area actually anesthetic. The root pains and the segmental or cord pains are frequently combined. Ordinarily the pain is lateral. It may occur first on one side, but, arising as it usually does from pressure upon the cord, it is evident from the anatomical situation that pressure and counterpressure must be equal and the whole diameter of the cord soon affected. When the tumor is within the cord and laterally situated, a partial Brown-Séquard palsy may develop at first, but ordinarily soon yields to the cross variety and the paraplegic syndrome. The pains are of all degrees of severity, but frequently atrocious in their intensity, of a lightening, darting, ripping character, with paroxysms and remissions. Exceptional cases run an entirely painless course.

The *anæsthesia* are so rarely sensitive that Starr,<sup>1</sup> in a masterly résumé of the subject, states that he never noted it in his own experience or in the literature. Pain over the seat of the tumor is also rare, and, when present, following the usual rule, is felt one or two inches below the level of the lesion.

The *reflexes* comport themselves as in the paraplegic state generally (see p. 373). Those which find their nuclear representation in the diseased segments are lost early. Those below the lesion are exaggerated as the compression is brought to bear, and, in extreme cases reach the highest degree of intensification. Should the cross-lesion of the cord become absolute, constituting a cord-division, all reflexes are lost below the affected level. The mental and visceral reflexes are subject to the ordinary rules of the paraplegic state. *Motor disturbance* of a paraplegic sort rarely precede the sensory disturbance, but usually follow it *pari passu* with the reflex manifestations. The feet and legs feel heavy, clumsy, and weak. This paresis becomes more and more pronounced as the pressure increases; the spastic gait is developed, and finally the patient becomes bedridden. Absolute motor loss is the great

<sup>1</sup> "Amer. Jour. Med. Sciences," June, 1895.



exception and depends, like complete reflex obliteration below the lesion, upon division of the cord.

With the motor loss, again, there is usually blunting of cutaneous sensation, and finally complete anesthesia, of paralytic distribution, surrounded by the hypersensitive band. Tumors in the cervical region may occasion cerebral symptoms: headache, choked disk, vomiting, and the hydrocephalic syndrome.

To again follow Starr, the order in which these symptoms arise is commonly: (1) Peculiar pains of limited distribution; (2) increase of reflexes below the lesion; (3) paraplegia; (4) loss of sensibility, and (5) loss of all subgenital reflexes. Bedsores and dystrophic joint-disturbance occur late in tumor, or may appear earlier upon the addition of an acute myelitis. Collins<sup>1</sup> names the clinical order of symptoms as follows: Sensory, motor, visceral, trophic, and topical; the topical consisting in tetaniform rigidities caused by pressure over the spine in the region of the tumor and in rare cases deformity of the spinal column.

Nonne<sup>2</sup> calls particular attention to a yellow color of the spinal fluid found in several cases of spinal cord tumor and to an increase of albumin in the fluid. Raven<sup>3</sup> has tabulated 47 cases in which increased albumin was noted without any cytological increase. He attributes the yellow color to blood-pigment. In some such instances the spinal fluid promptly coagulates in the test-tube. The nature and location of the tumor has no relation to the changes of the spinal fluid unless it by pressure closes off the dural sac.<sup>4</sup> Occasionally tumor cells have also been found.<sup>5</sup>

**Course and Prognosis.**—The course of the disease is usually slow from the insidious onset to the fatal termination by exhaustion due to pain, or the consequences of myelitis, cystitis, prostaticitis, bedsores, and septicemia. The rapidity of the disease depends upon the character of the growth, but tubercle is frequently of unexpected activity and may induce a complete paraplegia within a few weeks. In rare instances years have been consumed in the development of the tumor. The natural tendency is to paraplegia and death. The location of the tumor in the cervical or lumbar enlargement hastens the course of events.

The prognosis in tumor involving the cord, excepting gumma, is uniformly bad, and practically fatal without operation.

**Diagnosis.**—The diagnosis of cord-tumor depends mainly upon the insidious onset and the order of development of symptoms. The slow compression of the cord gradually induces the paralytic state with intense spasticity, but is preceded by the localized, persistent, and uniform root or segment pains. A *crise-aiguë* is ordinarily of sudden onset and not especially painful unless the meninges are affected. In that event the pain is diffuse and intense over the spine. Bedsores and trophic disturbance are early features of myelitis, late ones of tumor. The presence or history of new growth elsewhere aids the diagnosis, and the location in the dorsal cord is of some significance. In *Pott's disease* we soon have rigidity of the back, pain upon rotation or percussion or jars of the spine, and later vertebral thickenings and deform-

<sup>1</sup> N. Y. Med. Rev., Dec. 6, 1902.

<sup>2</sup> Deutsch. Zeitschr. f. Nervenk., Bd. 38, 1910.

<sup>3</sup> Ibid., Bd. 44, 1912.

<sup>4</sup> Ayer and Viets, Jour. Amer. Med. Assoc., Dec., 1910, p. 1707.

<sup>5</sup> Mohr, Deutsch. Zeitschr. f. Nervenk., Bd. 44, 1912, p. 418.

mities. The nerves are sensitive and the pain accurately follows the nerve-trunks as a rule.

The location of the tumor is deciphered by reference to the general rules of cord-localization (p. 347), especial importance attaching to the position of the early pains and the upper levels of dysesthesia. As already indicated, it is usually impossible to clinically determine whether a growth is vertebral, meningeal, or cordal, and operations are, therefore, to this extent exploratory. A Brown-Séquard paralysis, followed by a paraplegia, or analgesia preceding anesthesia, or dissociations of sensation, or limited early muscular atrophy, may indicate that the growth is within the cord.

Speculation as to the nature of the growth is based on those general features which are common to growths in all localities. The presence of malignant growths elsewhere points to a secondary neoplasm of the same order. Tuberculosis and syphilis have their own indications. It may also be borne in mind that sarcoma is the most common form of tumor in this location.

**Treatment.**—With the exception of gummata, which are to a considerable degree amenable to antisyphilitic treatment, the only course that promises relief is surgical operation. It is evident that tumor within the cord-substance can not be removed without great and permanent damage to the cord. It is also evident that if the compression from without has caused local cord-disintegration, an operation can at best only check the harm at that particular stage. Malignant neoplasms are very likely to recur, and are frequently secondary to large growths elsewhere that have already jeopardized life. Tubercle is also often secondary to a process that may leave little hope of prolonged existence. The fact, however, remains that without removal the cord-tumor itself will induce a fatality, preceded usually by the most intense suffering and the most abject helplessness. Even when an operation can do nothing but relieve the pains due to the local irritation, it may be favorably considered.

On the other hand, about seventy per cent. of recorded cases were operable, and out of thirty-three operated cases, tabulated by Pinman and Warren,<sup>1</sup> over one-half resulted successfully. Similar figures are given by Starr and also by Beuns.<sup>2</sup> Stursberg,<sup>3</sup> in a more recent and larger tabulation of reported cases, shows 32.2 per cent. of cures. Early diagnosis and prompt operation will materially improve the average.

#### SPINA BIFIDA.

Spina bifida is an embryological defect due to the failure of the neural canal to completely close in the posterior median line. As a result, the bony arches of the vertebrae are defective. This may be indicated by a depression, *spina bifida occulta*, or by a tumor made up of the contents of the spinal canal variously arranged. Its usual location is in the lumbosacral region, as the neural canal closes from above downward and is last completed at the caudal extremity. When present, it serves to fix the lower end of the cord to the corresponding vertebra, and the normal recession of the *conus medullaris* to the level of the second lumbar vertebra is prevented.

<sup>1</sup> "Am. Journ. Med. Sciences," Oct., 1899.

<sup>2</sup> "Die Geschwülste des Nerven-Systems."

<sup>3</sup> "Centralt. f. Grenzgebiete der Med. u. Chir.," 1906.



The simplest variety is the rare *meningocele*, which is usually covered by skin and consists of a pediculate or pedunculate sac of spinal meninges continuous with the arachnoid spaces. Neither the cord nor the nerves enter it, and deformities of the cord or disabilities in the legs and sphincters are absent.

The ordinary tumor in *spina bifida* is the *myelomeningocele*, made up of both the cord and the meninges and usually attended by deformities and paralysis in the legs and sphincters. The cord ordinarily is flattened out in the posterior surface of the tumor, which is more or less translucent, devoid of true skin, and only covered by a layer of epithelium. From this posterior situation the flattened and deformed cord-substance gives off the nerve-roots, which run forward through the sac into the intervertebral foramina. In the most prominent part of the tumor corresponding to the cutaneous defect and the location of the broadly spread cord-substance there is often a pit or umbilical depression.

In rare and more pronounced deformities the central canal of the cord dilates with the tumor formation, *hydromyelocele*, and lines its cavity, with which it is coextensive. The nerve-roots now lie in the sac-walls. Again, in *myelocele* a flattened mass—not a sac—of neural tissue lies in the vertebral hiatus, containing a small opening leading into the cerebrospinal canal. Through this, cerebrospinal fluid constantly issues. Both of these forms are rare, always attended by defects in the lower extremities, and the latter, *myelocele*, is promptly fatal in a few days. The occult form may only show a dimpling at the lower central sacral border, and this usually is still further masked by a nevus-like growth of hair.

**Etiology.**—The causation of this defect in the embryo, which dates back to the earliest days of gestation, like similar teratological defects, is obscure. It has been attributed to injury. In a number of instances it has appeared in several children in the same family and even in succeeding generations, showing hereditary transmission. Other children in the same family may present hare-lip or club-foot. It is more common in females than in males, in a ratio of about eight to seven.

**Symptoms.**—*Spina bifida* is usually discovered at birth, but may exist unnoticed as a slight depression, often covered by hair, for months or even up to adolescence, when the tumor or leg signs may appear. At first usually small in size, it may rapidly increase. When the cord is involved in the sac, there is usually associated club-foot or undeveloped legs or sphincteric incontinence. The tumor is in the middle line and presents varying appearances due to its make-up. The opening in the vertebral arches can usually be detected by touch, but the contents of the tense, fluctuating, and slightly compressible sac are rarely palpable. Under excitement and in the erect position the sac increases in tension and size. Pressure upon it is likely to produce uneasiness, stupor, coma, and convulsions. The fluid contents correspond to and are identical with cerebrospinal fluid.

The diagnosis is usually very easy, but it is more difficult to decide upon the exact nature of the tumor's makeup. The presence of deformities or paralysis in the limbs or of sphincteric incapacity and inability

cation would indicate that the tumor was not a simple meningocele, but that the cord was involved in the sac-wall. An osseous aperture and a nevus-like mass of pinkish tissue would suggest myelocele. The gray furnishes very definite evidence of any bony defect.

**Prognosis.**—Only in the slighter forms of *spina bifida* is the prognosis favorable. A pedunculated meningocele or mere depression is not incompatible with full vitality and perfect cord-functions. In the severer varieties, if the child lives, it will probably bear club-foot or other deformity and defect. The great majority of cases die during the first three months, cases of myelocele during the first three days. Rare cases have reached adult age and beyond.

**Treatment** is purely surgical. In meningocele a fair prospect is offered to extirpation of the sac. In the other and more common varieties, to extirpate the sac or to obliterate it by injections is to destroy a portion of the cord-substance. In this condition the sac must be preserved, and various osteoplastic operations have been suggested looking to the formation of a bony neural canal covered by cutaneous flaps. The deformities in the lower extremities and the paralytic condition, of course, would not be benefited thereby. In the occult form with late development of symptoms operation may also liberate the imprisoned nerves.

### CHAPTER III.

#### LESIONS PRINCIPALLY CONFINED TO THE GRAY MATTER OF THE CORD, AND DISEASES REFERABLE TO DISORDER OF THE SPINAL GRAY.

In this chapter a number of diseases are brought together that have in common symptoms dependent upon disorder of the lower motor neuron. In some, anatomical changes are clearly defined; in others there is reasonable presumption that eventually changes will be detected. The whole question of progressive muscular atrophy is in a transitional state, with a decided tendency to bring the various forms into close relationship.

##### ACUTE ANTERIOR POLIOMYELITIS.

*Poliomyelitis anterior acuta, acute spinal paralysis, acute atrophic paralysis, spinal paralysis of children, cerebral paralysis of children, infantile paralysis, poliomyelitis, Heine-Medin's disease, and epidemic paralysis* are the principal of the numerous names applied to an acute infective disease having a special selective action on the nervous system. It is marked by febrile and infectious symptoms, is of rapid onset and development, and promptly induces widespread muscular paralysis, a portion of which commonly remains permanently, and is then attended by local atrophy. It is often epidemic.



**Etiology.**—The great majority of cases occur before the tenth year, and fully three-fifths under four years of age, with especial frequency in the latter half of the first year. During the first six months it is extremely rare or else passes undetected to a fatal end. It has been freely attributed to *dehydration*, to *cold*, and to *traumatism*, especially in the form of falls, but it is doubtful whether any of these alleged causes are of direct importance. In many instances it occurs in the course of or during the convalescence from *infectious fevers*, especially the exanthemata. Its *infectious nature* is further indicated by the abrupt onset, the usual febrile movement, the gastric disturbance, the occasional occurrence of convulsions, and, most of all, by epidemic and endemic outbreaks. Such have been recorded by Gûnier,<sup>1</sup> Gûnlier,<sup>2</sup> Meûlin,<sup>3</sup> Leegard,<sup>4</sup> Odsolin, Nome, Calverly,<sup>5</sup> Altman,<sup>6</sup> Harbitz,<sup>7</sup> and the extensive epidemics in New York City in 1907, Melbourne in 1908, St. Paul, Minn., in 1909, Chicago, in 1917, by various reporters.

August, September, and October are the months of maximum epidemic prevalence in the northern hemisphere, the corresponding months below the equator, but sporadic cases may occur at any time, and frost does not immediately put an end to epidemics. In some of these outbreaks a considerable variation from the type has been noticed. Ascending myelitis, bulbar and pontine symptoms, encephalitis, cerebral ataxias, polyneuritis, meningitis, and even abortive forms are observed.

Landsteiner and Popper<sup>8</sup> were the first to cause the disease in monkeys by the intra-abdominal injection of a bacterium-free emulsion of the spinal cord of a lad who died from the disease. Similar experiments were made by Flexner, Strauss, and Hunsdon, and inoculation of monkeys remains the chief positive means of verifying and testing the infection. It does not matter how or where the virus is introduced into the circulation, but it is proved that the nasal mucous membrane furnishes a ready entrance and is probably the usual avenue of both infection and of elimination. Moreover, infection carriers are common among those brought into contact with the disease, as was shown by Petterson, who recovered the virus from the nasal and bowel washings of such persons. The early age incidence and the general adult immunity to the disease lead to the inference that such immunity must be the result of previous unrecognized infection. It is well known that many cases of poliomyelitis do not cause paralysis.<sup>9</sup> Flexner has practically proved that one attack produces permanent immunity. This rule may have exceptions. I have seen a case presenting one attack at the age of seven and a second at thirty-two. According to Flexner,<sup>10</sup> to whom most that is known of the infection is due, the virus can be regularly detected, both in man and monkey, in the nasal mucous membrane, brain, spinal cord, and the

<sup>1</sup>"Ann. Jour. Med. Sciences," 1843.

<sup>2</sup>"Hygiene," 1890.

<sup>3</sup>"N. Y. Med. Record," 1894.

<sup>4</sup>"Jour. A. M. A.," Oct. 20, 1907.

<sup>5</sup>"Zentralbl. für Bacteriologie u. exp. Therapie," 1909, ii, 377.

<sup>6</sup>"Erist. Bulletin No. 50, U. S. A. Public Health Service.

<sup>7</sup>"Jour. Amer. Med. Assoc.," Oct. 12, 1912.

<sup>8</sup>"Lyon Medico," 1888.

<sup>9</sup>"Neurolog. Centralblatt," 1890.

<sup>10</sup>"Austral. Med. Gaz.," 1897.

mesenteric lymph-nodes, but not in other bodily organs or structures or the blood. The virus thriving best in brain and cord structures and membranes eventually brings about anatomical changes in the nervous system with resultant paralysis. The period of incubation in monkeys may be two or three days or four or five weeks, apparently depending upon the amount and quality of the virus and the degree of bodily resistance. From the nasal mucous membrane, where the virus thrives, part of it is swallowed, leading to the mesenteric gland infections and contamination of the gastro-intestinal cavity and contents.

Aside from human carriers, it has been proved by Howard and Clark that bedbugs may so act, and Rosenau has shown that flies may transport the infection to monkeys either by contaminating their food or possibly by biting. All biting insects are under suspicion.

The virus is extremely minute, readily passing through the pores of the finest porcelain filters. In many ways it resembles the virus of rabies. Flexner and Lewis and Levaliti have produced an opalescence in bouillon cultures, in which Levaliti detected extremely minute bodies of oval shape. Römer and Joseph, by means of the ultramicroscope, found similar bodies in the Berkefeld filtrate. Flexner and Noguchi<sup>1</sup> have, however, discovered an extremely small globoid organism that will pass through a Berkefeld filter, propagates in ascitic fluid under anaerobic conditions, and produces poliomyelitis in monkeys, from which it can be recovered. It is also found by a special technic in the tissues of human cases and the experimental monkey cases.<sup>2</sup> It has been found in the blood by Amoss.<sup>3</sup>

Mather, Nuzum, and Herzog; Rosenow, Towne, and Wheeler<sup>4</sup> describe a coccus or diplococcus obtained from the brain, spinal cord, tonsils and abscesses in the tonsils which they believe to be the cause of poliomyelitis. Rosenow<sup>5</sup> further believes that this diplococcus by fusion produces the globoid bodies of Flexner, and has demonstrated such changes backward and forward by cultural modifications and inoculation determination. To these contentions the Rockefeller group reply that the coccus is a secondary or accidental invader.<sup>6</sup>

**Morbid Anatomy.**—The microscopical appearances, as well as the finer changes, vary greatly with the age of the lesion. The early descriptions based on recovered cases were misleading. At the end of one or two months a focus or several foci of myelitis are found in the anterior gray. There is local destruction of nerve-tissue, with dilatation and changes in the vessels. The ganglion-cells of the anterior horn involved in the lesion have lost their prolongations, show granular

<sup>1</sup> Jour. Exp. Med., Oct., 1911.

<sup>2</sup> Flexner and Noguchi, loc. cit.; Amoss, "Jour. Exp. Med.," vol. xx, No. 2, 1914.

<sup>3</sup> Mather's "Jour. Amer. Med. Assoc.," September 30, 1916, p. 1029; Rosenow, Towne, Wheeler, "Jour. Amer. Med. Assoc.," October 21, 1916, p. 1200; Nuzum, Herzog, "Jour. Amer. Med. Assoc.," October 21, 1916, p. 1205.

<sup>4</sup> Jour. Med. Research, March, 1917, p. 175.

<sup>5</sup> Bull. C. A., "Jour. Exper. Med.," 1917, p. 257.



disintegrations, or have entirely disappeared. The myelitic focus occupies one or both of the two arterial areas of the anterior horn; namely, that area supplied by the first branch of the anterior median artery (c



Fig. 141.—Diagram of arterial anastomosis producing infarctic paralysis (Chassagnon).



Fig. 142.—Section of spinal cord in a case of acute anterior poliomyelitis (Chassagnon). (c, c) Infarcted tissue not invaded by round cells (Williamson).

that field supplied by the arteries which enter the cord along the anterior roots, or both. Rarely it extends backward in the middle arterial field and invades the neck of the posterior horn, and usually it embraces the adjoining white matter of the cord to a varying extent. It is evident that the lesion is not strictly systematized, and it is equally evident that it is of vascular distribution. Batten<sup>1</sup> asserts, upon rather convincing evidence, that the vascular fault is primarily thrombotic, and that the lumbar cord is especially affected, owing to the circulatory disadvantages of the part. Wickman<sup>2</sup> does not accept the thrombotic explanation, and probably, if such takes place, it is of secondary occurrence.



Fig. 143.—1, Section of cervical cord in an old case of infantile paralysis. The right half is preserved in alcohol (Chassagnon). 2, Lumbar section in a case of infantile palsy; a, focus of old inflammation; left half of cord atrophied.



Findler, Harbitz, and Scheel,<sup>3</sup> in the Norwegian epidemic, found a patchy leptomeningitis more evident on the anterior aspect of the

<sup>1</sup> "Brain," autumn, 1904.

<sup>2</sup> "Handbuch der Neurologie," Bd. II, Lewandowski, Berlin, 1905.

<sup>3</sup> "Norsk. Mag. f. Lægevidensk." 1907.

cord and most severe over the areas of deeper-seated involvement, with great engorgement of the pia at these points. According to Fleiner and his associates the special globoid bacteria can be demonstrated in the foci of inflammation.

The myelitic foci vary in extent and number in various cases. Sometimes they extend through several segments of the cord eight or ten centimeters or more in a vertical direction, but usually are one to two centimeters in longitudinal extent. Sometimes single, more frequently there are several foci either on the same or on opposite sides of the cord and at various levels. They may invade the medulla, and the brain is similarly involved in some cases that perish within the first few weeks.

In cases of many years' standing the cord is seen to be shrunken on one side at the level of the lesion, and the cross-section is lacking in symmetry. The acute inflammatory condition has long disappeared, and a scar condition alone represents it. Nerve-fibers and cells are entirely destroyed. The absence of myelinated fibers renders the scarred area more or less glossy and translucent to the naked eye. The vessels are thickened and appear more numerous on that account. The shrinking of the lateral half of the cross-section involves mainly the gray matter and especially that of the anterior horn, but the white columns and the posterior cornu may also share in it to some extent. If the original lesion is sufficiently extensive to involve the spinal apparatus of an entire extremity, or even



Fig. 140.—Case of poliomyelitis affecting the lower extremities bilaterally; foot-drag on both sides. Note the widening of the right leg and thigh.



Fig. 141.—Case of poliomyelitis affecting right arm (years), right arm, and shoulder.

of a segment of it, changes in the cord above the lesion, and in the brain, are found analogous to those induced by amputations.

The motoric roots arising from the injured part of the cord are often diminished in volume and may or may not show alteration of structure. This is true equally of the mixed *nerve-trunks*. The exact condition depends apparently upon the age of the lesion and the complete or partial disappearance of the peripheral prolongations of the spinal



pyramidal cells that have been injured. In the paralyzed *muscles* we have the same changes that are found after division of the nerve-trunk, but a few muscular fibers often persist, some of which may even show hypertrophy. The rest of the muscle is fibrous and fatty. Infiltration of fat sometimes reaches a condition that may properly be designated lipomatous. The *bones* in the paralytic members are retarded in their growth, and in old cases dating from childhood are undersized in comparison with those of the sound side. They lack the sharply defined muscular markings, and the Haversian systems are found to be undersized and poorly developed.

**Symptoms.** Usually in sporadic cases, without apparent cause, the child is found to be feverish and ill. A temperature of 100° to 102° F. has been frequently noted, and this febrile invasion stage lasts from a few hours to several days, when *paralysis* and *flexibility* of one or more limbs are detected. It is not rare, however, for the child to go to bed apparently well and to awake paralyzed in the morning. The febrile movement may be attended by vomiting and diarrhea, by convulsions of a generalized character, or by *delirium* and diffuse cerebral manifestations. As soon as the paralysis is noted, the case is usually recognized.

Most writers state that there is a complaint of pain in the afflicted members only rarely, but that, as a rule, *sensation* in all its phases is entirely normal. It is probable, however, that early dysesthesia, owing to the usual infantile age of the patient and a lack of careful search for such difficulty, has been frequently overlooked. In some considerable number of cases handling of the affected limbs during the initial fever provokes *urticaria* which are not elicited by similar manipulation of the other members. Complaints of pain and of formication have been generally noted in older children and in adults, lending, perhaps, undue weight to the usual supposition that such cases are not of a true spinal type. In some cases there is even severe pain.

In recent American epidemics a somewhat modified clinical picture has been more common. This consists in a more distinct febrile period; prominence of hyperesthesia; slight rigidity of the neck with pain on forward traction of the head; and even a slight development of Kernig's sign. Muscular tremors, weakness of the lower extremities, headache, nausea, vomiting, and constipation or diarrhea have been early symptoms.<sup>1</sup> The *spinal fluid* frequently is turbid and then upon shaking in the test-tube produces a persistent froth or foam. More commonly it is clear. There almost invariably is a pleocytosis of mononuclear lymphocytes, an increased amount of globulin and albumin, and a positive reduction of Fehling's solution.

The *sphincters* are seldom relaxed, so that control of the bladder and bowel remains unimpaired, but in the rare cases in which the sphincters are relaxed there is more or less apparent loss of sensation, the extent of the lesion is greater, and the prognosis is extremely unfavorable.

Even in fat children the implicated *nerves* can be seen, after a few weeks, to have wasted, and if tested with the faradic current, either do not respond at all or show a remarkable diminution in their excitability. At this time the patient will have begun to show considerable improve-

<sup>1</sup> A. Ziegler, "Jour. Amer. Med. Assoc.," March 17, 1917, p. 818.

ment, the motor paralysis remaining complete only in the parts that are to suffer permanently, and a gradual improvement may be expected to extend over several months. In the muscles showing lessened faradic excitability galvanism commonly produces exaggerated responses, as compared with the sound limbs, and the complete reaction of degeneration or any modification of it may be encountered. In a well-marked case faradism fails by the tenth day, and the increased galvanic response appears, lasting for about six months, when it gradually declines. At this point faradic excitability returns and the muscle regains something of its size and strength; or, if too seriously impaired, faradic response does not reappear, galvanic response disappears, and the muscle is irretrievably lost.



Fig. 142.—Deformity of foot after lumbar poliomyelitis acuta.



Fig. 143.—Deformity of upper extremity and thorax after poliomyelitis anterior acuta.

The *reflexes* are lessened or abolished in proportion to the implication of the cornual cells making up a part of their arc. *Bones* which have not attained their full growth are frequently retarded or entirely fail to develop if their trophic centers are implicated.

The seriously atrophied muscles become contracting fibrous bands, and, since they offer neither assistance nor opposition to the synergic and ant synergic muscles, *distortions* soon develop, with *joint-changes* and sometimes *subluxations*. Joints which depend upon muscular support, as the shoulder, may allow of so much deformity by the relaxation of the muscles which have lost their tonicity that the articular surfaces widely separate. The skin is inactive, often cold, and sometimes dry and scaly, but the atrophic conditions so usual in neuritis are practically absent and bed-sores are almost unknown.



The lower extremities are affected about three times as frequently as the upper, and the left leg twice as often as the right. A crossed form, in which the upper extremity on one side is involved with the opposite lower limb, is not rare; but involvement of both limbs on the same side is extremely uncommon. In the *lower extremity* the extensors seem more susceptible than the flexors; hence drop-foot, with talipes equinus, flexed knee, and flexed thigh are common. When the paralysis is below the knee, the sural muscles usually escape. In the *upper extremity* the most frequently encountered wasting is in the small muscles of the hands, the deltoid, and the extensors of the wrists. The biceps and supinators generally escape.

The distribution of the paralysis of muscles follows their segmental relations in the cord, and, consequently, when limited is of a functional character. The very fact of such functional distribution at once incriminates the cord. The muscles of the trunk are in rare cases totally and widely affected, producing weakness in the back, deviation of the spinal column, and defects in the muscles of the chest. Ordinarily, however, the segments of the cervical and lumbar enlargements are selected. A small focus of anterior poliomyelitis in the dorsal region hardly produces local signs, as the trunk and thoracic muscles are represented in a considerable vertical extent of the cord. In rare cases, however, the abdominal or chest muscles or those along the spine are involved very decidedly. In a less degree this is not rare, as shown by spinal twists, protuberant abdomen, and abnormal respiratory movements of the thorax.

The bulbar muscles do not always escape, as is shown by various ophthalmoplegias, squints, facial and hypoglossal palsies, which are now and then recorded, and would be more often seen did not coincident pneumogastric accidents usually terminate such cases fatally.

**Course and Forms.**—The course of the disease, after a period of incubation that may be as short as two days or as long as two weeks,<sup>1</sup> may be clinically divided into: (1) A stage of febrile invasion, lasting from a few hours to several days, with local tenderness and rapidly developing and increasing paralysis; (2) a stationary stage, lasting for several weeks; (3) a period of improvement, lasting to the end of a year, and (4) a stage of permanent disability for the remainder of life. Relapses during the early weeks have been recorded in rare instances. Second attacks are extremely rare. Among the sequelae the contractures, dislocations, and deformities have been already mentioned. The fragility of the bones makes them liable to fracture, but union takes place with ordinary promptness under proper fixation.

The *adult form* is in no way different from the infantile cases, excepting the variations due to complete growth having been attained. In monolateral cases the onset of the disease is insidious and the course may be subacute. After infectious diseases one or more limbs may be found useless, flaccid, and atrophic, declaring the antecedent myelitis. Occasionally the onset is marked by pains of a severe, cutting character, which are frequently attributed to neuritis or to rheumatism, but may be due to the irritation of the sensory pathway in the cord by the location

<sup>1</sup> S. Fleissner, "Jour. Amer. Med. Assoc.," July 22, 1910, p. 279.

of the myelitic focus backward, involving the neck of the posterior horn, or to an accompanying meningitis.

An ascending form, presenting the clinical picture of a Landry's paralysis, is observed in some instances— in almost every epidemic outbreak, and many of the cases of so-called Landry's paralysis are undoubtedly instances of poliomyelitis presenting an upward invasion course.<sup>1</sup>

It has been noted by a number of observers that in rare instances, years after the acute disease has subsided, a slow, insidious *compensatio* has led to a progressive muscular atrophy, which declares itself first in the limbs that were originally paralyzed by the acute process. This may progressively invade the entire muscular system.

**Diagnosis.**—In the early stages of fever, before paralysis has appeared, diagnosis usually fails except under epidemic conditions. The termination of an apparently trivial ailment in extensive paralysis is frequently the cause of much chagrin on the part of the medical attendant, who may naturally enough have expressed a favorable prognosis. As already indicated, the initial fever may be readily mistaken for that of general disorders, and sometimes, though rarely, the pain in the limbs leads to the idea of rheumatism. If, however, the possibility of anterior poliomyelitis be in mind, and examination discloses some slight local tenderness or diminished muscular activity, or both, a guarded opinion will naturally follow. It is only when the paralysis is developed or developing that the nature of the disease becomes certain, and even then, if there have been cerebral symptoms, such as delirium or convulsions, difficulties are not at an end. The cerebral palsy of children is almost invariably ushered in by convulsions, but these have a definite distribution involving one side or one limb, or only the face, while the convulsions of the disease under consideration are generalized. Localized pain, from *traumatism* or inflammation, may cause immobility of a limb, and when preceded by fever gives rise to a doubt; but the usual absence of extreme sensory disturbances in disease of the anterior horns is a distinguishing feature, while the local conditions are usually marked by active muscular resistance. After a few days the electrical test gives absolute data. Faradic response is abolished in no other disease so early; even in severe neuritis it is longer maintained, and is then attended by a very marked sensory disturbance, which also usually precedes it for a long time. A few careful applications of the induced current at this period can do no harm. The progressive infantile myopathies are of extremely slow development and are symmetrical in distribution. Spinal puncture in the early stage yields a fluid containing the evidence of inflammation in lymphocytes, turbidity, increased globulin and albumin, and increased reducing activity to Fehling's solution.

**Prognosis.**—So far as life is concerned, in sporadic cases this disease terminates fatally only exceptionally, and if the patient survives the onset of the paralysis but a short time, life may be considered out of danger. In recent epidemics, however, in this country the mortality has reached 20 and even 40 per cent. The danger is in direct relation to the involvement of the medullary functions, and is indicated

<sup>1</sup> Basse, "Transactions Chicago Path. Soc.," Dec., 1908.



by the presence of cerebral and cranial nerve symptoms. Further, in non-fatal cases, one may say with a reasonable degree of certainty that the paralysis at first developed will notably recede, but, unfortunately, it is almost equally certain that a portion of it will permanently remain. At the end of a fortnight a carefully conducted faradic examination of the muscles enables the physician to speak more specifically regarding the amount of permanent disability. At that time any muscle which responds, however feebly, may be expected to regain a fair degree of its former tone and strength. Even after several months repeated faradic stimulation of a muscle, at first perfectly inactive, may develop some contractility, and this is of favorable import for the given muscle. The extent of permanent paralysis governs the amount of resulting contracture and deformity; and, likewise, the retardation of development of the limb and of the bones is in similar relation. The anticipated amount of these deforming conditions will have a bearing on the probable general activity of the individual, his prospective physical health, and liability to fractures. The possibility of the late appearance of a progressive muscular wasting may be kept in mind.

**Treatment.**—Every case should be treated as one of infectious or contagious character, with such measures to prevent dissemination as are usually employed in infectious and notifiable diseases. All persons in intimate contact with a case of poliomyelitis should be considered capable of acting as carriers. The systematic use of antiseptic nose and throat sprays by all such should be required.

Owing to the irregularities of the course of this disease in various cases, and its natural tendency to improve up to a certain point, it becomes a matter of great difficulty to estimate the value of any therapeutic agent or mode of treatment. In the early stage, as soon as the diagnosis is made, if fever still continues, there is good reason to suppose that antipyretic antiseptics, like the salicylates, large doses of the tincture of the chlorid of iron, or even bichlorid of mercury, would be good. The free use of urotropin is strongly recommended in the hope of producing an antiseptic effect in the spinal fluid and nerve tissues. Hot applications can be made to the spine if the circumstances of the patient will insure their intelligent and faithful employment; otherwise they are worse than useless. The child should be kept on the side or face, and the affected limbs should be thoroughly enveloped in cotton-wool to maintain the circulation and the nourishment of the muscles in the parts laboring under diminished trophic influence. There is hope that a specific antitoxin may be soon available. Treatment with the blood-serum of those who have had an attack has not yielded the hoped-for results, though both immunity and curative results in the early stages of the disease in monkeys have been attained by Fleener. Abrahamson has also successfully immunized monkeys. The use of antistreptococcus serum has some advocates. The use of spinal stimulants like strychnin, while the lesion is active, is to be avoided; but when the active process has come to a standstill—that is, ordinarily, at the end of a fortnight—its systematic use is one of the most important measures. The *acrid-*

ness of electricity consists of maintaining the nourishment and normal contractility of the muscles, which are temporarily deprived of their natural motor and trophic control, so that, as the inflammation subsides and the wide-spread inhibitory effect of the local lesion recedes, the central apparatus may find the muscular periphery in the most favorable state to respond to its enfeebled influence. For this purpose, as the faradic response is early abolished, the interrupted galvanic current must be used, the slightest intensity being employed that will cause a contraction. Care must be taken not to unduly fatigue the muscles. A dozen contractions at most should be elicited at one sance, and often only one or two can be provoked by any strength of current that is bearable. Care not to alarm the child is imperative, as a daily struggle will do more harm, probably, than the electricity will do good. As the muscles often react better and with less pain to the positive pole than to the negative, it is well to have for the negative electrode a broad sponge which may be placed on the sacrum or breast, and with a smaller, positive sponge the muscles may be exercised. Applications of galvanism through the cord are quite useless, and even if such currents reached the lesion, which is doubtful, their effect for good is questionable.

Later on, as faradic response returns in the muscles only slightly affected or temporarily inhibited, this form of electricity is efficacious for the purpose of local stimulation, and the presence of this reaction in any muscle is always, as already indicated, a gratifying circumstance. To intrust galvanic electrical treatment to the parents, however intelligent they may be, is a mistake.

In the same way local frictions and salt baths, warm wrappings, and massage are valuable measures which can be more rationally trusted to parents or nurses who take an intelligent interest in the work. They may be gently employed from the first day, and when electricity is not tolerated or can not be systematically employed, must be relied upon to replace it. The moment a group of muscles weakens, the limb tends to assume an abnormal position, and it is very important to meet this tendency from the very first moment, even in cases where there is every probability that the paresis will recede. It can be easily accomplished by means of the warm wrappings, or even by the application of light splints. There can be no question that recovering muscles will find their task much easier if their proper relations have been maintained. Unbalanced muscles will be much less liable to contractures if an artificial balance has been provided and joint-surfaces have not been altered by long-maintained vicious positions.

As soon as the permanent paralysis can be fairly well foretold, massage should be especially directed to obviate the contractures and deformities that ordinarily result. Stretching of the unopposed muscles by passive movements of the joints will accomplish much, and the moment a tendency to contracture is perceived, the case becomes one for mechanical appliances. The tendency to talipes equinus, for instance, can be met by a light elastic cord from the toe of the shoe to a band at the knee; and more elaborate orthopedic apparatus should be employed at the knee and hip if required. Two important points should guide



the treatment of the paralyzed parts: first, never to permanently immobilize any joint or muscle that has a particle of voluntary power; second, to persist with gentle massage, light supports, and re-educational exercises if need be for years. From first to last local measures must be gentle. Atrophic muscles can be destroyed by too vigorous massage, and electrical stimulation and active and passive movements can be easily overdone.<sup>1</sup>

Nearly all the improvement that is to take place in the muscles will have developed by the end of the first year, and what is slowly subsequently gained is usually dependent upon continued massage, exercises, and similar local measures.

The treatment of a late or neglected case is practically surgical. Shortened tendons may be cut and joints straightened. Tendons of healthy muscles may sometimes be transplanted to take the place of those paralyzed. Much is being accomplished by nerve suture. The involved nerves are being grafted into sound ones and the innervation of the atrophic area re-established. A resection at the knee is sometimes of advantage to secure a rigid limb instead of a useless contortion or a dangle-leg. By using a high shoe or other appliances crutches may often be laid aside.

### SYRINGOMYELIA.

The central canal of the cord is sometimes enlarged through congenital defect. This may be a part of a general ventricular distention, causing hydrocephalus and spina bidda. In certain instances attended by any symptoms during life a tubular cord has been found post mortem. These cases present *hydromyelia* and are of tentological origin. The term *syringomyelia* is now limited to acquired enlargement of the central canal or to the formation of entirely new canals of considerable length in the gray substance of the cord. Such canals are the result of gliomatous infiltration about the central canal or in the gray horns of the cord and its subsequent degeneration, forming tubular, cystic cavities. In rare instances a central myelitis or a central hemorrhage may cause a fusiform or tubular excavation that can not always be clinically distinguished from the neoplastic variety or may in turn induce the latter. Some cases probably originate in spinal hemorrhage occurring at birth. This disease, considered rare in the nineties, is now frequently recognized, and in neurological clinics furnishes about the same percentage of patients as infantile cerebral palsy.

**Etiology.**—Men are much more frequently affected by this disease than women, and especially men exposed to hard labor. Verhagen and Vanderschelde report several instances of syringomyelia in the same family, but a neuropathic heredity is rare. Adult years furnish nearly all the reported cases, but it has been observed well developed at nineteen by Langdon, of Cincinnati, by the author at sixteen, and by Ballard and

<sup>1</sup>Lowry and Martin, "Jour. Amer. Med. Assoc.," March 4, 1916, p. 729.

Thomas at three.<sup>1</sup> Cerebral rheumatism, exposure to bad weather, transudation, overwork, the acute infectious fevers, and syphilis,<sup>2</sup> have been reported as possibly causative in various cases, but this relation, in the gliomatous form, at least, is entirely conjectural.

**Morbid Anatomy.**—The syringomyelic cord in marked cases shows notable changes of conformation that correspond to its tubular condi-



FIG. 14.—Sections of a syringomyelic cord. 1, Lower thoracic region; 2, upper thoracic region; 3, cervicobulbar region (Bridg).



FIGS. 14a and 14b.—Sections of two syringomyelic cords showing common locations and extent of cavities (Bridg).

tion. It is soft, flattened, and sometimes fluctuating. The collapse of the canal may produce a furrow, so that the cord seems doubly. The condition finds its favorite locality in the cervical region, but may extend throughout the entire length of the cord or be confined to any portion of it. Its upward extension carries it into the fourth ventricle. According to Haenel,<sup>3</sup> medullary participation is found in about one-third of all cases.

The cross-section usually reveals a single oval cavity, or there may be several in communication or independent. The customary situation

<sup>1</sup> "Am. Jour. Med. Sci.," March, 1899. <sup>2</sup> Nisslthaus, "Zett. f. Nervenk.," Feb., 1893.

<sup>3</sup> Lewinsky, "Handbuch der Neurologie," Band II, S. 384, 1911.



is in the immediate neighborhood of the central canal and behind it, but it may extend laterally into the anterior or more commonly into the posterior horn of gray matter, either on both sides or unilaterally. Again, it may be limited to one posterior horn. While the white columns of the cord may be spared, it is not unusual for the glimitous process to invade them, especially the pyramidal tract, when the gray matter has been widely destroyed and the cavity has attained large proportions.

The cavity contains cerebrospinal fluid, which is sometimes bloody or gelatinous. Its walls are made up of a well-defined substance, outside of which the cord appears infiltrated, dense, and even sclerosed. In the medulla, when the lesion extends so high, the gray matter is first similarly affected.

The microscope shows the usual new formation to be glimitous and rich in blood-vessels, especially on the external periphery. The predominance of various elements gives rise to varieties such as pure glioma, neuroglioma, and vascular glioma, all of which may be present in the same cord. Schultze describes an infiltrating sort as a gliosis. It does not usually result in cavity formation, and is, therefore, distinguished from the glimitous variety. He is supported by Hoffman, and Holt and Harter<sup>1</sup> report a case. Orłowski<sup>2</sup> has seen a double canal, one arising from dilatation of the central canal and the other from gliosis and degeneration outside the central canal. Charcot, Hillebrand, Joffroy, and Armand insist that in some cases the initial step is a central myelitis, which Schultze, Dejerine, and others deny. The nerve-tissues involved in these various processes are strangled and destroyed even before the formation of cavities. The cells first yield their prolongations and then disappear. The axis-cylinders outlast the myelin. The meninges and spinal roots are usually unaffected except in the form associated with cerebral pachymeningitis. Destruction of the anterior horns is followed by the usual trophic disturbance in the associated periphery, such as wasted muscles, perforating ulcers, and cutaneous dystrophy. The interference with the cells of Clarke's columns is supposed to account for the joint-lesions that are commonly present, and which are identical with the arthropathies of locomotor ataxia.

**Symptoms.**—From the nature of the lesion it is apparent that the symptoms may in different cases embrace perversion and obliteration of all the various functions of the cord. Nevertheless, their peculiarities and groupings are sufficiently distinct to frequently enable a diagnosis. As there is no common type, the symptoms must be arranged somewhat arbitrarily.



FIG. 345.—Case of syringomyelia with areas of paraneurotic dermatitis marked in black. There is cervical syphilis.

<sup>1</sup> "Ann. Jour. Med. Sci.," April, 1895.

<sup>2</sup> "Arch. de Neurol.," Sept., 1908.

**Sensory Disturbances.**—The early involvement of the sensory pathways in the gray commissure and in the posterior horns and columns produces related sensory symptoms which are well-nigh characteristic. There are usually definite practically coextensive areas of thermo-anesthesia and analgesia, with retention of the tactile sensibility. This is sometimes denominated the *sympathetic dissociation of cutaneous sensation*, and is highly important to the diagnosis. The patient fails to distinguish the temperature of water or objects brought in contact



FIG. 146.—Various distributions of thermo-anesthesia and analgesia (Bruns).

with the skin, though recognizing contact, and may receive burns in this way without experiencing any pain. The thermo-anesthesia may be complete or partial, and is then relative to the part examined and somewhat to the patient's intelligence (see Part I). Sometimes heat is felt as pain, or "hot" as "cold," and all possible variations of degree, and confusion may be encountered, including well-marked anesthesia dolorosa. The distribution of the thermo-anesthesia is also significant. Rarely it may be hemiplegic; it is seldom general. Ordinarily, it involves the limbs, or portions of them, such as would be covered by a glove or sleeve, a sock or long stocking. It may be limited to thoracic or abdominal bands or stripes along the limbs. It may be unilateral or symmetrical. In figure 147 it is of unusual outline, but will be noticed to roughly conform to some spinal segments. In some cases areas at first discrete have been observed to coalesce into anatomical cord-territories. The mucous surfaces are similarly affected.



FIG. 147.—Thermo-anesthesia and analgesia (Bruns).

The analgesia may be partial or complete, and usually conforms in outline in a general way to the thermo-anesthetic area, or it may be more extensive. It embraces all the tissues, so that felons, caries of bone, and disintegrating joints may be painless and insensitive.

As a general rule, *haptic* or *epicritic* sensibility is perfect. A slight diminution of it is not inconsistent with the diagnosis, and its obliteration follows extreme invasion of the posterior columns, being then a



part of the absolute anesthesia that marks such a lesion. An anesthetic area may thus have a border of thermo-anesthesia and analgesia, or these conditions may be found in different parts of the body, in relation to the amount and distribution of the cord disease. In some cases and at early stages pains of a tabetic, neuralgic, or burning sort are persistent. Other patients complain of a constant warm or burning sensation in a given region. As a general rule, deep and protopathic sensory features are more involved than the superficial or epicritic. Thus, sensitiveness of joints, of the testicle, and eyeball is often blunted or abolished.

**Motor disturbances** are dependent largely upon the invasion of the anterior horns and the pyramidal tracts. They are, therefore, usually secondary in point of time to the sensory symptoms with which they come to be associated. As the anterior horn is invaded, the associated muscles lose power and their reflexes diminish and disappear. Some muscular atrophy is almost always present, and it may reach a high grade. Its distribution depends upon the part of the cord involved, and may conform to many of the typical spinal atrophies, such as the form of Duchenne-Aran, for which it has no doubt been frequently mistaken. It may be progressive or advance by spurts, and is most common in the upper extremities. Fibrillary twitching and reaction of degeneration mark the atrophic muscles, as in other muscular atrophies of spinal origin, and their strength is reduced in proportion to the atrophy. When the gliomatous process invades the lateral tract, spastic features are induced. These may be associated with disorders due to involvement of the anterior horn, producing the conditions found in amyotrophic lateral sclerosis, but the face is usually unaffected. Involvement of the posterior columns causes ataxia and incoordination. Tremors, cramps, fibrillation, choreoid movements, and various muscular twichings are not infrequent.

**Trophic Features.**—The affected extremities, in addition to the muscular atrophy, often present trophic disturbances, especially about the digits, similar to those in neuritis. Glossy skin, hypertrophic nails, increased or diminished perspiration, and herpetic and bullous eruptions are encountered. Cuts, burns, and abrasions heal badly or tend to permanent ulceration. In this way paronychia causes the nails to fall. Fekou is rather common, especially in the Moroccan type, and causes mutilations of the fingers by the loss of several phalanges, unattended, usually, by the slightest pain. Perforating ulcer is encoun-



Fig. 150.—Case of syringomyelia, showing atrophy very right scapula and thorax and in right leg. Sympathetic.

tended with some frequency. Boils, abscesses, and other local infections are not rare. They all heal badly, produce extensive scars, and may cause mutilations and deformities. A thickening and hardening of the skin, especially of the fingers, is common, and variations of Raynaud's syndrome are often added.

The arthropathies are almost invariably represented. They affect the spine by preference, and the articulations of the upper extremities more frequently than those of the lower limbs. In some cases the bones are affected. They are fragile, readily fractured, and unite with difficulty and with persisting callous deformities. In a few cases the hands have been enlarged, as in acromegaly, for which this disease has been mistaken. They have even been found associated. The spinal arthropathies give rise to deviations of the vertebral column in over one-half of the cases. Usually it is a scoliosis, but angular deformities are not infrequent. The muscular weakness may account for some of the spinal deformities. A deep cavity at the upper portion of the chest in front has been noted several times by Astie<sup>1</sup> and Schröder has reported two cases presenting habitual dislocation of the shoulder due to changed contours of the humeral head and the glenoid cavity.

Vasomotor symptoms are represented by dermatographia, blanching or redness, edemata, and localized disturbance of perspiration, especially in the affected areas.

Unusual Symptoms.—In various rare instances the following unusual and rather accidental conditions have been present, due to the special location of the glomerular disease in the particular cases: Loss of sphincter control, sexual impotence, suppression of menstruation, papillary inequality, narrowing of the palpebral fissure and retraction of the eyelid, nystagmus, facial paralysis, hypoglossal paralysis, optic neuritis, pneumogastric accidents, glycosuria, polyuria, and pronounced bullar invasion, producing a progressive bullar ulcer and other cranial-nerve disabilities. Such cases are sometimes designated *syringobulbia* (Fig. 152).

Course.—Syringomyelia is a chronic malady of slow progression and final termination. It often presents stationary periods



Fig. 152.—Hand and foot in case of syringomyelia of Marfan's type, showing, 1, mutilation of the finger from atrophy, 2, atrophy and paralysis of the finger, and 3, bullar invasion.

or even slight temporary improvement may be noticed. Bullar invasion means early termination. It has exceeded forty years' duration in favorable cases, and may be interrupted by death from concurrent or

<sup>1</sup>"*Thèse de Paris*," 1897.



accidental disease. Its logical termination is in death by exhaustion or by bulbar crises. Ulcerations, dystrophic conditions, or infections from such sources may induce a fatal termination.



FIG. 134. a.—Charcot-Marie-Tooth in syringomyelia.

**Clinical Forms.**—The ordinary form has been in view in the preceding lines. One clinical variety is furnished by *Morvan's disease*. In



FIG. 151. b.—Illustration of *morvan* in Fig. 134. a.

this form the sensory dissociation is marked, especially in the hands and arms, with associated atrophy and paresis. There are mutilations of the fingers by successive whitlows and marked cutaneous dystrophy. Sordosis and arthropathies are usually present. Zamboni insisted that this is a form of leprosy, and Prus<sup>1</sup> demonstrated in such a case a bacillus closely resembling that of leprosy. On the other hand, Joffroy and Achard have demonstrated central cavities in Morvan's disease, and this is confirmed by Thomas.<sup>2</sup> The leprosy idea can no longer be entertained. In both a peripheral neuritis is common, and Prus suggested that the propagation of the leprous bacilli in the cord might furnish the irritation that incites the gliomatosis.

There is more than an accidental relation between syringomyelia and a cervical *hypertrophic purpurumyelia*. Brossard<sup>3</sup> insists that the meningeal process usually precedes and furnishes the irritation that sets

<sup>1</sup> *Leçons sur les Maladies Nerveuses*, 1895.

<sup>2</sup> *Archiv. f. Psychiatr.*, Bd. xxvii.

<sup>3</sup> *Ber. Méd. de la Suisse Romande*.

up the central gliomatosis. It seems probable that any permanent cord irritation may lead to a secondary syringomyelia.

**Prognosis.**—The outlook in any given case is ultimately fatal, but the duration of the disease must be estimated by the course it is running and the portions of the cord affected. Bulbar symptoms are extremely grave and lumbar involvement is hardly less so. The tendency to stationary periods and slight remission must, however, be kept in mind.



Fig. 112.—A case of syringomyelia with involvement of the lower extremities.



Fig. 113.—Syringomyelia with involvement of the lower extremities.

On the other hand, hemorrhage in the gliomatous area is rather common and causes initial symptoms, serious complications, and fatal effects.

**Diagnosis.**—The diagnosis depends upon the insidious development of the disease and upon the combination of sensory, trophic, and motor disorders. The dissociation of touch and pain is well-nigh distinctive, but is occasionally found in tabes, neuritis, and hysteria. Usually it will be necessary to exclude progressive muscular atrophy, amyotrophic lateral sclerosis, pachymeningitis hypertrophica cervicalis, Pott's disease, cervical ribs, locomotor ataxia, and peripheral neuritis. Perhaps acromyagia, scleroderma, leprosy, and hysteria may at times confuse, but in all these an attentive study of the combinations of symptoms should enable a diagnosis to be made.

**Treatment** has proved futile, or nearly so. Measures looking to the general well-being of the patient are most advisable. Locally, massage and electricity may give slight help at times. Counterirritation over the spine must be used with caution, as it is capable of producing extensive and rebellious ulceration. It is possible that x-ray applied along the spine in the region of the gliosis might retard the neoplastic activity, and some favorable cases have been reported. In the same way radium might be employed. When it is possible to diagnose a pachymeningitis



or to localize the gliosis in a limited vertical cord area, surgery holds some promise of amelioration. It is probable that some of the intramedullary spinal tumors that have been operated upon, and occasionally with success, are of this character. One operation done for me in a well-marked case of cervical syringomyelia resulted in the removal of the intramedullary growth and very great improvement.

The patient must be warned to avoid the ever-present danger of burns and infections, against which he is no longer guarded by normal sensibility. Muscular strain may precipitate hemorrhage into the gliomatous tissue, with very serious or fatal results. For the neuralgic pains and occasional cramps various analgesic and quieting remedies may be employed, but with preference for the milder and habit-free drugs. Warm baths and hot applications usually answer the purpose.

#### PROGRESSIVE MUSCULAR ATROPHIES PRESENTING LESIONS OF THE SPINAL GRAY MATTER.

All progressive muscular atrophies may be divided into those in which (1) lesions of the spinal gray matter are found, and (2) those in which no such changes are discernible by our present means of investigation. It may at once be admitted that this division is arbitrary and probably temporary. It is not unlikely that many members of the second group will eventually be found in the first. The present conception of the integral character of the lower motor neuron, embracing the spinal pyramidal cell, its peripheral prolongation, and the muscular organ under its motor and trophic dominion, is inconsistent with the idea that the so-called muscular dystrophies or idiopathic muscular atrophies are independent of disturbance in the spinal gray. For purposes of convenience they will be separately described, though every production is found from first to last, and even a single case may present several of the varieties at one or in successive periods.

Duchenne, followed closely by Aran, in 1849 and 1850 described a progressive muscular wasting without sensory disturbance, which they called progressive muscular atrophy and thought it a disease of the muscles. Cruveilhier, in 1855, recognized it as a spinal disease, and Lockhart Clarke first limited the lesion to the anterior spinal gray matter. Charcot still further differentiated the lesion and pointed out its dependence upon degeneration of the ganglion-cells of the anterior horn. Later on Charcot distinguished a variety of progressive muscular atrophy in which the lateral tracts were also degenerated, and called it *myotrophic lateral sclerosis*. The first variety is now called by French writers *Duchenne-Aran's disease*, the second, *Charcot's disease*. Subsequently, some writers, notably Marie, denied the existence of the Duchenne-Aran type in toto. Others, as Gowers, insisted that both the Duchenne-Aran type and the type of Charcot are identical, only varying as the lateral tracts or the anterior gray—that is, as the upper or lower motor neurons—are first or most degenerated. Gowers stated that he had never seen a case in which lesions in both spinal regions could not be detected, and cases presenting lesions confined absolutely to the anterior gray no longer appear in current literature. Many cases

formerly classed with the Duchenne-Aran type were doubtless those of multiple neuritis, syringomyelia, Charcot's disease, and the so-called idiopathic muscular atrophies to be later described. More recently Hoffman,<sup>1</sup> Eulenbergh, Ducloux,<sup>2</sup> Dufrenoy, and Long<sup>3</sup> have reported cases clinically indistinguishable from the spinal type of Duchenne, but presenting neuritic lesions only. We may, therefore, properly discard many of these conflicting terms and speak of progressive muscular atrophy with or without cord lesions.

Attention is now directed to the first sort, which embraces conditions variously called *spinal progressive muscular atrophy*, *wasting palsy*, *chronic poliomyelitis*, *syringomyelic lateral sclerosis*, *atrophia muscularis progressiva spinalis*, *Duchenne's disease*, and *Charcot's disease*.

**Etiology.**—Progressive spinal muscular atrophy is practically a disease of adult life occurring between the ages of twenty-five and fifty, but



FIG. 181. Normal motor root. FIG. 182. Section from a case of atrophic spinal sclerosis. (MAYO.)

It may appear earlier or later. It is somewhat more frequent in men than in women. Direct inheritance is very rare, but a neuropathic tendency can occasionally be traced. It has been noted as following various alleged vague causes, such as anxiety, overwork, exposure to cold, concussion of the spine, syphilis, and various infectious and septicemic states. Its appearance years after an acute poliomyelitis has been already indicated under that caption. A close inquiry will sometimes detect a history of symptoms antedating the alleged cause. There can be little doubt that at least in some instances it is an expression of toxicological defect in the motor and trophic portions of the central apparatus, and constitutes a primordial shortcoming by which these parts reach an early death. Since the Wassermann and spinal fluid tests have been available, positive findings in these cases indicate that syphilis is a more common feature, if not an actual cause, than was formerly supposed.

**Morbid Anatomy.**—The lesions of progressive spinal muscular atrophy embrace in rare cases the entire motor field of the nervous apparatus from cerebral cortex to muscular nerve-endings, and include

<sup>1</sup> *Neurolog. Centralbl.*, 7, 1899.

<sup>2</sup> *Nouv. Ann. de la Salpêtr.*, 1, 1902.



the muscles themselves. Both upper and lower motor neurons in their entirety are destroyed by a degenerative process. Following the pathological rule that a neuron degenerating from toxic cause or involution first shows changes in its peripheral portion, the upper motor segment may present alteration only in the pyramidal fibers of the cord. This may reach the medulla, and, as a rule, does not extend into the pons, medulla, capsule, and cortex, though it may do so. In the lower neuron the degeneration is probably at first peripheral, but in all cases that reach a marked development the cells of the anterior gray are found degener-

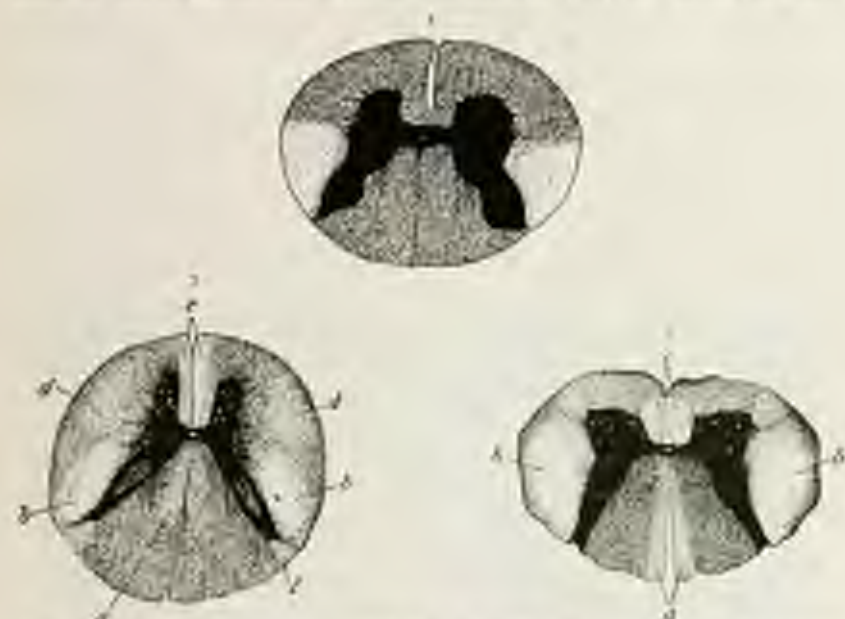


Fig. 163.—Atrophies in a case of amyotrophic lateral sclerosis. 1, lumbar region; 2, dorsal region; 3, cervical region (Mann).

ated. Attending this we have muscular atrophy, with fibril and fatty changes and degeneration in the motor fibers of the nerve-trunks, limited sharply by the anatomical relations of the diseased components.

In the *vent* the gray substance of the anterior horns shows atrophy. The ganglion-cells, many of which usually have disappeared, are wasted and degenerated, and there is a general shrinking of all the nervous elements of the horn. The *gray substance* of both the direct and crossed pyramidal tracts shows sclerotic degeneration. This process is not strictly confined to them, but usually involves the anterolateral tracts to a lesser degree, and may invade the lateral limiting layer. This is especially the case in the upper dorsal and cervical regions. The columns of Goll sometimes show slight changes, apparently due to the shrinking of the myelin, and not to an active degeneration. The lesions of the anterior cornua are generally most pronounced above the dorsal region, but the involvement of the crossed pyramidal tracts extends to the lowest cord-levels. Similar changes may be traced through the *medulla*, both

in the gray matter below the fourth ventricle and in the pyramidal tracts above the decussations. These latter may ascend through the *poliuscles* and *internal capsules* to the pyramidal-cell layers of the cortex, and there a cellular degeneration, identical with that in the spinal gray, may occasionally be encountered.

The *muscles* are pale and fatty, and under the microscope present various changes. The fibers may be narrowed, with striae poorly marked or less frequent than in health, or more frequent and deeply marked, showing a tendency to disintegration. The striation may entirely disappear in fatty granulations, and distinct globules or the empty sarcoplasmic sheaths may alone remain. Longitudinal striation sometimes develops, and the transverse markings may later disappear. The *nerve-trunks* often contain degenerated fibers, which can be traced through the anterior spinal roots to the anterior horns. All these central and peripheral changes vary in degree in different cases, and intermediate instances are becoming more and more frequently noted in which the muscles or the nerve-endings or the anterior cornu cells show preponderating changes. Only the most apparent tache can be relied upon to determine minor abnormalities.



Fig. 26.—Hand in advanced case of amyotrophic lateral sclerosis, showing muscular wasting and the characteristic spastic position of thumb.

**Symptoms.**—The various types of spinal muscular atrophies and the confusion that has arisen over them, as well as the symptoms in various cases, are referable to the vertical extent of the lesions and the varying intensity of the process at different levels. It must at once be evident that if the degeneration falls first on the lower neuron, flaccid, atonic, atrophic paralysis will appear in the periphery. On the other hand, if the upper neuron be primarily affected, the paralytic state will be spastic and the tonic atrophy will be marked by rigidity. All degrees between these extremes are encountered in practice. Flaccidity may be present in the upper and spasticity in the lower extremities in the same case. Involvement of the upper neuron can only be evidenced through the lower in the way of increased reflexes and spasticity. If, then, the lower neuron leads the upper in the degenerative



course, the latter can not produce its signs. Again, the degenerative process falls by preference on the cervical cord and manifests itself in the upper extremities first. Thence it tends to extend, and eventually reaches the cranial nuclei in the bulb, inducing a progressive bulbar palsy. In other cases the higher levels are first affected, presenting bulbar palsy or ophthalmoplegia, and the downward extension of the disease may be cut short at any point by pneumogastric accidents. Labiodyspharyngeal paralysis or bulbar palsy is identically the same disease first affecting the bulbar centers. It is also variously combined with the spinal forms. (See page 157.)



FIG. 361.—A case of progressive muscular atrophy of the spinal type in the fourth year of the disease. All skeletal tissues affected.

The muscular atrophy and weakness usually come on together and progress equally. In the great majority of cases wasting appears first in the radial half of the hands, sometimes first in one hand, sometimes in both hands at once. In another group of cases the shoulder-muscles are first affected. In the hands the disease ordinarily affects first the thenar muscles. It, therefore, appears to elect those muscles which show the highest differentiation of function and which represent the latest motor requirements in the evolutionary scale,—namely, circumduction of the upper limb and the opposition of the thumb to the fingers. If the morbid process be one of involution, we might naturally expect such a program. It results in flattening the palm, and the thumb falls back into the same plane with the other digits, producing

the "ape hand" (Fig. 156). The interossei and lumbricals are also affected, and again most on the radial side of the hand. Furrows between the metacarpals mark the loss of the small muscles, and the integument hangs loose and redundant with numerous folds and wrinkles. The bones are entirely denuded of muscular covering in severe cases. A persistence of subcutaneous fat sometimes obscures the muscular atrophy. In late cases there is a marked tendency to clenching of the fingers, from the involvement of the small muscles, so that the first phalanges are extended, the second and third sharply flexed (Fig. 156). They may be rigidly and spastically fixed, all vol-



Fig. 156. Hand in amyotrophic lateral sclerosis, showing atrophy of the interosseous and lumbrical muscles of the fingers and thumb.

untary motion destroyed, and passive motion much limited. The muscles of the forearm suffer next, or they may escape for a time and the shoulder-muscles waste first. In other cases the muscles of the neck are first involved, and then those of the upper extremity, or the wasting may even develop first in the leg and buttocks. Muscles like the pectoralis, deltoid, and trapezius, which have different associations, and receive their innervation from different spinal levels, may waste in corresponding portions. It thus happens that the upper portion of the trapezius is frequently spared until late in the disease, as it is controlled by the spinal accessory. Again, it may suffer early. The muscles of the back are among those early involved, and those of the scapula are commonly first affected, allowing corresponding displacements of this bone and limiting the use of the arm. The thoracic muscles are also involved, impairing respiration, and if the abdominal groups suffer, breathing may become purely diaphragmatic. Wasting in the legs is much less common and is usually less in degree, though atrophy may here first



shoot itself. In the same way the face escapes for a time, or the disease may commence in the bulbar nuclei, and labioglossopharyngeal palsy may be the first step toward generalized progressive muscular atrophy. Finally, no voluntary muscle may escape.

The muscular wasting and loss of power result in corresponding changes of contour and position deformities. The patient may be unable to hold the head erect. The uncovered acromion renders the shoulder angular. The claw-hand, the scoliotic spine, the displaced scapula, and the distorted chest manifest the unsupported skeleton. The characteristic of the atrophy is its gradual invasion of a muscle fiber by fiber, commonly preceded by fibrillary twitchings. These twitchings are sometimes decidedly exaggerated, causing rhythmic jerking movements of the hands and arms or twitchings of facial muscles.

The reflexes may be either abolished or more commonly increased in activity. Early and rapid wasting is often marked by diminution and even extinction of the reflexes in the affected muscle; but, again, there is often early rigidity and marked myotatic irritability, which last throughout the disease and persist even when the muscles are much wasted; or reflexes at first exaggerated may disappear. The reason for this variability has been already indicated.

In the lower extremities, however, it is the rule that the reflexes are augmented and spastic disturbance of a paraplegic sort is commonly found unless the atrophy begins in the lower members. This gives rise to the spastic gait often marked by clonus and dragging footsteps. The rigidity may show itself by the fixity of position, even when the patient is seated or in bed. The legs tend to extension, the arms to semiflexion, and the hands are held in the negative position, midway between pronation and supination. The quadriceps are seldom affected. An excessive jaw-jerk can sometimes be elicited, and implies bulbar extension of the disease. The cutaneous reflexes are normal so long as their muscles survive the atrophic invasion.

The bulbar symptoms of the disease correspond to those of bulbar palsy (see p. 157). Fibrillary twitchings about the mouth and eyes and in the tongue are followed by wasting, and the characteristic facial expression is developed. In advanced cases the open mouth, the drooling saliva, the difficulty of deglutition, the nasal voice, and the pneumogastric pulsies only too plainly indicate the upward extension of the disease and the critical condition of the patient. The pharyngeal reflex usually persists as long as the pharyngeal muscles have power to act. The mind is not disturbed.

The electrical response in nerves and muscles is, as a rule, quantitatively reduced for both currents, and finally extinguished. In a rough way it is proportionate to the amount of muscular fiber present. In most instances the reaction of degeneration is found in a few muscles, or any variation of it may be presented.

Sensibility is practically intact. In some instances there is complaint of dull pains at or before the onset of atrophy, and vague feelings of weakness, fullness, and formication may be mentioned during the disease. The paralytic and wasted limbs are usually cold and the circulation is

poor, but trophic disturbance in the skin, perforating ulcers, arthropathies, and bed-sores are unknown. The visceral functions are not notably impaired.

**Varieties.**—The chief varieties to be distinguished are: First, cases marked by flaccid, atonic atrophy, which reaches an extensive degree, usually first appearing in the small muscles of the hands,—the Duchenne-Aran type; second, cases marked by similar wasting, but less in degree, and preserving tonicity, rigidity, and increased reflexes from the first,—myotrophic lateral sclerosis, or Charcot's disease; third, an intermediate variety with only slight muscular wasting, but with great weakness and with spasms and retained reflexes; fourth, the variety beginning as a laléoglossopharyngeal paralysis, an ophthalmoplegia, or some bullar palsy.

**Course.**—These spinal muscular atrophies may commence in various ways and first involve any portion of the cord or the bulb. The clinical course varies accordingly. It is, however, progressive, and may terminate in a few months or a year, or consume twenty or thirty years in its evolution. Apparently stationary periods in the protracted cases are not uncommon, but the logical termination in all is death from cardiac or respiratory failure. Intercurrent disease, especially acute infections and particularly pneumonia, are badly borne and frequently end life.

**Diagnosis.**—In fully developed cases of the spinal progressive atrophies the diagnosis is usually not difficult, but a differentiation from the so-called *idiopathic muscular atrophy* of the next section may sometimes be impossible. We rely upon the presence of fibrillary twitching, increased reflexes, if only in the legs, and the reaction of degeneration in at least some of the muscles, to indicate the spinal location of gross changes. These cases usually begin in adult life and show no particular family history; the second group usually begin in childhood and are frequently familial. The spinal varieties select the muscles of the hands, shoulders, or hips first; the idiopathic varieties commonly spare the hands at first and may select the humeral muscles or the peroneal group first, and are frequently marked by pseudohypertrophies, especially of the calf, gluteal, and scapular muscles. *Multiple neuritis* ordinarily affects all four extremities and can be traced to some competent infection or poisoning. In it the sensory symptoms are prominent from the first, while they are slight or absent in spinal myopathies. *Syringomyelia* may produce a local atrophy, but has its distinctive index in the dissociation of cutaneous sensations and the usual presence of arthropathies and other trophic disorders, besides usually being unsymmetrical. *Multiple arthritis* is sometimes marked by extreme muscular atrophy. Here we have the early history of articular disease and the continued existence of arthritic mischief. The wasting, too, is usually on the proximal side of the joints, thus sparing the small muscles of the fingers. It is as likely to appear in the lower as the upper extremities, is rarely accurately bilateral, and affects first and principally the joint extensors. The reflexes may be increased, if not inhibited by pain or ankylosis, and the faradic irritability is usually increased. *Transverse myelitis* is limited distinctly



by an upper border of sensory and anatomically corresponding motor disturbances. The onset and clinical history are acute.

**Prognosis.**—The prognosis is always grave, and in proportion as the disease tends to invade the bulb, death is imminent. Intercurrent affections present more than their proper danger.

**Treatment** is practically futile, but should none the less be conscientiously instituted. Any trace of syphilis should be heretically treated. The continuous use of nitrate of strychnia, as recommended by Gowers, in increasing dose, for long periods of time by the hypodermic method should be faithfully tried. Beginning with  $\frac{1}{16}$  grain, three times a day, the dose may be gradually increased, under proper supervision, to  $\frac{1}{8}$  grain at a dose and even to  $\frac{1}{4}$  grain in some cases. The application of the thermocautery to the back, the careful use of exercises, massage, and electricity to the muscles, and endless attention to the general health and the processes of nutrition and elimination may prolong life. The danger of choking to death or of inducing an aspiration pneumonia when the pharynx is involved must be borne in mind. It is to be hoped that some product, perhaps of biochemistry, may be discovered that will maintain vitality in the degenerating motor apparatus and postpone the involutional changes of the disease.

#### PROGRESSIVE MUSCULAR ATROPHIES NOT MARKED BY STRIKING HISTOLOGICAL CORD LESIONS.

The view-point regarding a group of clinical forms that have in common the feature of progressive muscular weakness and atrophy, often associated with hypertrophy or pseudohypertrophy of some muscles, has altered markedly during the past few years. When it was first found that such cases presented no spinal lesion, they were termed *progressive muscular dystrophies* by Erb, and *peripheral progressive myopathies* by Charcot. A variety of forms were described by different observers, whose names, unfortunately, became associated with and served to fix these variations, which are now, following Erb, Brisson, Sachs, and others, grouped under a common head. Writers also refer to the *pseudohypertrophic* form, the *brachial*, *scapulohumeral*, *pelvic*, and *peroneal* types, as hypertrophy prevails or the atrophy is most pronounced in the various indicated regions. As material has accumulated, transition forms have been encountered with increasing frequency. Two or more of the foregoing types have been found in the same patient or in members of the same family. What is more important, the same family has presented cases of progressive muscular atrophy of the spinal form and also of the so-called idiopathic muscular variety in one or in succeeding generations. Again, there is an increasing number of observations going to show that in the so-called myopathic cases the spinal gray is not absolutely normal. A much larger series of observations discover changes in the muscle nerve-endings or in the peripheral extremities of the lower neurons. The so-called peroneal or atrophic type furnishes an intermediate form between the spinal and the alleged purely muscular varieties. This is clinically indicated by the fibrillar twitchings, the reaction of degenera-

tion, the relation of the muscular dystrophy to certain spinal segments, and the functional relation of the affected muscles. Yet similar segmental and functional outlines are presented by all cases. This alone compels an acknowledgment of the spinal factor. An attentive and intelligent study of any given case will usually show the features of several of the so-called types and serve to prove their essential identity. The upper spinal levels or the musculature pertaining to the upper spinal segments are first invaded in some; in others, the lower portion of the cord first manifests the disease.

If any significance is attached to the atrophy of the lower motor and trophic neuron, it will be impossible to consider the cell-body in the spinal horn as above reproach when its axonal prolongation shows degenerative changes, or when its trophic control is perverted or destroyed. At present such change in the cell may be called dynamic or functional when no morphological alteration can be discovered, but the pertinent fact remains that actual changes appear in some instances, and the probability is strong that improved technique will discover them in all. Degeneration, here as elsewhere, is not allowable, and the very interesting reports of Leeson<sup>1</sup> and Petren<sup>2</sup> on anencephalic and anucleic conditions, attended by full muscular development, prove at least an embryonic interdependence between muscle and nerve-cell. Sainton<sup>3</sup> has found well-marked changes in the spinal cord in the neuritic form, and Alodie and Denoyer,<sup>4</sup> in a typical pseudohypertrophic case, obtained the electrical reactions of degeneration that are supposed to be a part of spinal and nerve disintegration. Indeed, as before insisted, every gradation, from progressive spinal muscular atrophy to cases only showing muscular changes, can be adduced. Some observers, however, insist that the spinal changes are secondary to the muscular atrophy,<sup>5</sup> and well-examined cases still fail, in some instances, to present any demonstrable cord variation, even when the clinical type suggests the spinal variety of disease.

These muscular atrophies, to adopt that term for convenience only, have in common a familial tendency. They often appear in several members of a given family or in blood-relations of the same or different generations. While they may appear at any age, they show a marked tendency to occur in the early years of life, and commonly affect the roots of the extremities rather than their distal ends. They are almost always unmarked by changes of sensibility, by fibrillar twitchings, and by the electrical reactions of degeneration.

**Etiology.**—Regarding the causation of these progressive myopathic atrophies very little can be positively stated. Their appearance in several members of the same family or in several members of succeeding generations, their interchangability of form, their propagation by the females, their appearance during the age of active growth and at developmental epochs of life, stamp them as hereditary, familial, and embryonic. Meck has demonstrated that new-born animals possess many more muscle fibers than adults, as, for instance, in the biceps, and Pick<sup>6</sup>

<sup>1</sup> *Nerv. Syst. in Schist.*, June, 1899.      <sup>2</sup> *Ibid.*, Aug., 1900.

<sup>3</sup> *Neurolog. Centralbl.*, 12, 8, 223.

<sup>4</sup> *Virchow's Archiv.*, Bl. 131, S. 438.

<sup>5</sup> Pick, *Deutsch. Zeit. f. Nervenhe.*, Bl. xvi, 1900.

<sup>6</sup> Pick, *loc. cit.*



suggests that in some instances this natural disappearance of muscle fibers may, from prenatal or other cause, exceed physiological limits and produce progressive atrophy. The flabby and wasted muscles of old age may be in part due to a similar cause. Age.—The great majority of cases appear before puberty is established, with especial frequency during the second dentition and at pubescence. Others are first manifest during adolescence, from eighteen to twenty-six years of age; and a rapidly diminishing series is encountered later in life. It sometimes happens that, appearing at adult years in one generation, the myopathy follows in early childhood in the next. As males are rendered impotent by the disease, its transmission necessarily falls to those females of the family who escape and reach maturity. In addition, boys seem



Figs. 133 and 134.—Spondylomedullary system, progressive muscular atrophy. Note the angle on right side of neck with hunching of the neck, due to wasting of the trapezius, and the position of the pelvis.

more frequently affected than girls. Appearing commonly during the years of childhood, it often follows the diseases peculiar to that period of life, but it is difficult to assert their causative activity. In some cases diphtheria has seemed to lead to it; in others it has followed exposures to cold, slight injuries, and infections. Given a nervous fiber endowed with enduring qualities, it is not impossible that any condition capable of reducing the general health, or any toxic state may set upon it with unusual violence.

**Morbid Anatomy.**—The muscles show various morphological changes, depending upon the presence of hypertrophy, pseudohypertrophy, or atrophy,—the final and logical end for all the affected muscles. The sarcolemmal elements may be hypertrophied or atrophied. Enlarged fibers may be found in a mass of greatly wasted muscular tissue. There

is round-cell infiltration, vacuolation, splitting, division, and longitudinal striation of muscle-fibers, with hyperplasia of connective tissue and an increase in fat that may reach the highest degree of lipomatosis. The early changes consist of increase of connective tissue, possibly some muscular hypertrophy, then muscle atrophy and fatty infiltration. What appears clinically as a large muscular mass, in some instances is found to be a markedly lipomatous structure almost devoid of muscular fibers.

Von Bakes<sup>1</sup> has found the muscle nerve-poles undeveloped and degenerated. Horsley, Strumpell, and Marinesco have described the peripheral nerves as degenerated in the peroneal or leg type of the disease; the muscles and the anterior pyramidal cells of the cord were also involved. Hoppe has found similar changes in the facioscapular-humeral variety. Sachs and Brooks<sup>2</sup> have observed universal shrinkage of the posterior root ganglion cells in one case. To ordinary reagents the nerves and pyramidal cells usually react in a normal manner, but the finer technique of recent years is likely to determine changes in both.



Fig. 142 and 143.—Case of facioscapular humeral type, showing facies, rigid mouth, lordosis (Horsley, etc.).

**Symptoms.**—The most striking symptoms depend upon the change of nature in muscles and their loss of strength. These are variously combined in different cases. We will first take them up in systematic order, and finally group them in describing the common types of the disease. The myopathic facies depends upon the paresis of the facial muscles. The face presents a various, sleepy, inert expression, and fails to adequately show forth the lively emotions that may actuate the

<sup>1</sup> La Semaine Méd., Aug., 1901. <sup>2</sup> Amer. Jour. Med. Sciences, July, 1901.



patient. The brow is smooth, and the frontalis is unable to assist in raising the thin and drooping eyelids. The wasting of the eyelid muscles may give the eyeball a false appearance of prominence and cause a haggard expression. The lower lid, from the laxity of its tissues, often tends to droop. The cheeks and lips are flaccid, and the lower portion of the face droops from the bones. The eyes can not be firmly closed nor the lips puckered. In some cases the lips are thickened by hypertrophy and drag down, increasing the nasolabial furrows. The mouth may then remain open constantly, with a thickened, pendulous lower lip. In other cases the lips are thinned, motionless, and can not be retracted from the teeth. In either case puffing, whistling, or spitting is poorly performed or impossible. The movements of the eyeballs are usually not disturbed, but in two cases the author has noted a very great loss of conjugate lateral movements. Movements of the tongue may also be greatly limited. The palatal excursions may likewise be reduced in amplitude, and laryngeal disturbance may be added.



Fig. 162.—Progressive muscular atrophy at shoulder girdle, showing deformation about shoulders, with pseudohypertrophy of trapezioid, wasting of all dorsal muscles, and spinal index in vertebrae.

Beissel calls particular attention to an apparent lengthening of the neck that he finds a constant symptom, and due to the drooping of the shoulder. The direction of the *oblique* is frequently outward and downward, instead of upward, as in health. This may be accentuated by the early involvement of the trapezius, causing the characteristic angle, as shown in figures 159 and 160. The drooping shoulder may also appear only on one side. Attempts to extend the arms laterally, or even to hold them rigidly at the sides, cause a widening of the neck by the prominence of the upper borders of the trapezioid. The *suprascapular hollows* are thereby inordinately exaggerated and the sternomastoids spring into great prominence. Even the upper inner angle of the scapula may become salient above the normally curved line from mastoid to acromion. The scapula is usually mobile, and may wing out freely from the chest. The *squati* muscles, especially the infraspinati, have a marked tendency to pseudohypertrophy, which may add greatly to the scapular

deformity and accentuates the anterior curve of the lumbar spine when viewed from the side. This curvature is very commonly present, and may reach extreme proportions. It is due to the weakening of the spinal erectors in part, but is increased greatly when the glutei allow the pelvis to tip forward on the heads of the femurs, and is dragged into still greater prominence by the protruberant belly, due to the weakened abdominal muscles.

The deltoid requires particular mention. It is commonly hypertrophied and shows the marked discrepancy between the bellies and extremities often found in long muscles. The fibrous changes are marked at the origin of the muscle from the scapula and clavicle, apparently displacing the full, round shoulder-cap to the outside of the neck of the humerus, where it stands out as a lumpy mass (see Figs. 159 to 174). Similar changes in contour are occasionally encountered in the biceps of the arm, in the anterior crural, and in the sternal group. The forearms and hands are commonly spared, at least until very late in the disease, but K. Meusel<sup>1</sup> has reported a case showing early involvement of the interosseal muscles of the hands. The glutei and the gastrocnemii are favorite locations for pseudohypertrophy. The calf-muscles also are frequently shortened so that the patient can not raise the toes from the floor while standing on his heels nor can the foot be passively flexed beyond a right angle. This interferes with the gait, causing the patients to stumble over the smallest obstacles. It may even produce an equinovarus. In rare cases a general increase in the adipose tissue may greatly obscure the underlying muscular defects and maintain a fatigued appearance of muscular development that is belied by great weakness. In the same way the muscles presenting pseudohypertrophy look immensely strong, and feel very firm, but are found lacking in contractile power and subject to fibroid shortening.



Fig. 164.—Showing the atrophic and parietic condition of the affected muscles and of the contractures that sometimes appear in them.

The attitude presented by the patients is the result of the atrophic and parietic condition of the affected muscles and of the contractures that sometimes appear in them. It, therefore, varies with the location of the disease. Owing to the shortening of the calf muscles, little difficulty is found in maintaining the ankle-joint at a proper angle unless an equine position of the foot has developed, raising the patients to stand upon their toes. Standing then becomes extremely difficult. The position at the knee and hip is often entirely dependent upon the ligamentous support of these joints. This allows the pelvis to tip forward upon the head of the femurs and necessitates a strong anterior spinal bend to carry the head and shoulders back into the line of gravity for the maintenance of the equilibrium. The prominence of the abdomen is thus produced and is accentuated in turn by the weakness of the abdomi-

<sup>1</sup> "Neural Central," July 1, 1902.



nal muscles. In some cases the head is tilted backward, but when the spinal curve is pronounced the chin may rest on the sternum. Ordinarily the feet are kept wide apart to increase the base of support.



Figs. 165, 166, and 167.—Waddling gait in a case of pseudoparalytic paralysis.

The gait is broad-based and waddling (Figs. 165, 166, and 167). The foot to be advanced is clumsily put forward, the pelvis tilting sharply downward on that side, the body being inclined over the supporting foot as a counterpoise to the swinging limb. This is repeated in the opposite sense at the next step, and the patient advances in a waddling, waddling, awkward fashion, stumbling over the least obstacle, and falling heavily if the very unstable equilibrium is momentarily lost. Mounting stairs is often an early difficulty.

The manner of rising from the ground is most characteristic. If placed on the back, the patient may have great difficulty in rising at all. His usual plan is to turn over on his face, then huddle himself together, and get upon his knees. The usually weakened psoas, vastus, and gluteal muscles are now inadequate to the effort of lifting him. He, therefore, advances the body into the "all fours" position, and, carrying the weight of the head and shoulders on the arms, pushes up the lower end of the trunk with his legs as a cow gets up by the hindquarters. The hands are now brought toward the feet, one is placed above the knee on the same side, then the other at a higher point on the thigh of its own side; the shoulders and head are pushed upward, the pelvis tilts forward, the sway-back suddenly appears, and the patient attains the erect attitude by a process of climbing up his own legs (Figs. 168 to 173). In late stages standing and walking may be impossible.

It is needless to say that in the cases in which the legs and pelvic muscles escape or are only slightly affected, the attitude, gait, and manner of rising are not disturbed. The sway-back may also disappear when the patient is sitting, or may then give place to a rounding of the back, the patient resting his elbows on his knees or otherwise gaining a forcible support for the upper part of the trunk.

The upper extremity is most affected by the impairment of the mus-

cles of the shoulder-girdle. Next in frequency the brachial group is affected, while the muscles of the forearm and hand are usually spared. In the *lower extremity* wasting rarely avoids the gluteal and psoas groups



Figs. 164, 165, and 166.—Method of raising from the ground in cases of paraplegia.



Figs. 167, 168, and 169.—Method of raising from the ground in paraplegia.

and commonly affects the anterior crural distribution. The calf-muscle, with the glutei furnish favorite locations for pseudohypertrophy, while the calf-muscles in addition are the commonest of all locations for fibroid contractions, which may, however, appear in any muscle in



the late stages of atrophic shrinking. The small muscles of the foot may escape. The peronei are selected first in the leg type.

The *trachinotomus* are usually diminished, and when wasting or pseudohypertrophy is apparent they are commonly abolished. The ordinary case shows only a quantitative reduction of electrical muscular excitability for both currents. In rare instances, and usually in the peroneal or so-called neuritic type, the reaction of degeneration may be obtained. Fibrillary twitchings and severe pains are equally rare, but are found under similar circumstances. The *apoplexics* are not affected. Sensation is intact in all its modes and senses. A large proportion of these cases, and perhaps all of them dating from early childhood, show



FIG. 124.—Marking atrophy, wasted and well defined in a case of pseudohypertrophic paralysis.



FIG. 125.—Contracted muscles, peroneal type, as in case marked by pseudohypertrophy. (From "Osteitis deformans, atrophic paralysis, and pseudohypertrophy," by Karl and Vossler.)

some *mental backwardness* and apathy. Trophic disturbances are not encountered, but growth may be retarded and the bones may be diminutive.

**COURSE.**—These conditions are progressive. The muscular wasting usually steadily advances, with its attendant and increasing weakness. Some cases present stationary periods without assignable cause, and resume their course without adequate explanation. As a rule, the younger the age at which the disease appears, the more rapidly does it incapacitate the individual. It commonly occurs in girls at a later period than in boys, and then runs a slower course. When commencing in childhood, sometimes an age of forty years may be reached, but death usually occurs between twenty and thirty. The disease is not, as

\* Schatz, "Deutsch. Zeit. f. Nervenheilk.," April, 1899.

a rule, fatal in itself, except by pneumogastric accidents; death ordinarily occurs from *intercurrent* disease.

**Varieties.**—A number of clinical varieties have been described and are worthy of some attention, but it should be borne in mind that they are not distinct morbid conditions.

The earliest recognized form is the one called *pseudohypertrophic paralytic*. It usually appears in early childhood, affects boys much more frequently than girls, and is marked by extreme enlargement of the calves and buttocks. These stand out in intensified relief next the wasted thighs and forward-tilted pelvis. The psoas group is early affected, rendering going up-stairs difficult at an early date. False hypertrophy is also commonly found on the dorsum of the scapula, in

the lower border of the pectoralis, and the lower part of the deltoids. The serratus magnus, erector spinae, and humeral muscles commonly suffer early. The myopathic facies is not well marked, but can be detected in late cases. The forearms, hands, and feet are commonly spared until the last. The enlarged muscles finally shrink, and present all the features of those that shrink from the first.

The *peroneal or leg variety*, sometimes called the *Charcot-Marie type*, or the *neurotic form*, is marked by the early atrophic shrinking of the peroneal muscles. The calves are next involved and the thighs follow. The disease may thus be confined to the lower extremities for years. Eventually it invades the trunk and upper extremities, first affecting the hands. It shows fibrillar twitchings, the electrical changes of degeneration, and presents undoubted cord-lesions.

The *juvenile type of Erb*, also called the *brachial form*, affects mainly the muscles of the arms and shoulders,

and appears in early youth, and usually in several members of the same family.

The *facioscapulo-humeral form*, or the *type of Landouzy-Déjérine*, affects, face, shoulder, and arm, or may be considered as the brachial form plus facial involvement. These cases furnish marked instances of the myopathic facies.

The *infantile or Werdnig-Hoffmann type* occurs in family groups, usually appearing during the first years of life and coming to a fatal termination



Fig. 111.—A mixed case showing facioscapulo-humeral involvement, peroneal wasting, and a foot deformity similar to that of Friedreich's disease. There were fibrillar twitchings and the reaction of degeneration in the leg muscles.



in one to four years. The movements and control of the hip-joints are first affected, followed by feebleness of the muscles of the back and abdomen, then of the neck and shoulders. Later the atrophy and paralysis affect the arms and forearms, so that the child is rendered quite helpless. The facial, buccal, and facial parts are not implicated. Death ensues from respiratory feebleness and pulmonary complications.



Figs. 175, 176, and 177.—Case of the anterior variety of family progressive atrophy.

Oppenheim, in 1900, described a condition of congenital amyotonia (*amyotonia congenita*) which shows the same condition of the muscles as the family myopathies, but rarely presents family or hereditary traits. To distinguish it from Thomsen's disease, Haberman<sup>1</sup> proposes the name congenital atonic pseudoparalysis. It is congenital or may suddenly appear after infections. The reflexes are lost from the first, tactile sensibility is commonly greatly reduced, the distribution of muscular weakness spares the distal portions of the limbs, and there is little or no tendency for it to spread. Some cases recover, and most show some improvement. Two postmortem examinations by Ranby and Collie re-

<sup>1</sup> *Ann. Jour. Med. Sci.*, Mar., 1903.

spectively showed<sup>1</sup> atrophy of portions of the anterior horns and nerve roots; one by Spiller showed no such changes.

Several of these forms may be present in the same patient, as the leg type with the facioscapulohumeral variety, or pseudohypertrophy in the legs and the brachial type above. The hypertrophic variety regularly becomes shrunken finally, and lumps of false hypertrophy, on the other hand, may appear in muscular groups otherwise greatly wasted.

**Prognosis.**—The outlook is always unfavorable, but some of these myotrophies appear late in life, and do not apparently shorten it. The menace of the disease falls upon the respiratory apparatus and heart. Intercurrent diseases also find an easy prey in the weakened organism.

**Treatment.**—Unfortunately, all forms of medical treatment here given negative results. Carefully selected exercises, graduated to the capacity of the weakened muscles, promise the most good. Gowers early insisted upon this, and Wiener<sup>2</sup> has recorded a very encouraging instance in which much improvement was secured. Massage to the wasting muscles may have a beneficial effect. The application of electricity is usually rendered impossible by the painfulness of the currents required to actuate the muscles if much wasted, and it is not certain that its vigorous use is harmless. General measures pertaining to the health, comfort, and education of these unfortunates will be suggested by their individual requirements. Sometimes a tendency of the feet to turn is needed to keep them on their feet, but the use of sustaining corsets and braces usually augments the muscular feebleness by depriving the parts of their only natural exercise.

#### ARTHRITIC MUSCULAR ATROPHY.

All varieties of joint-disease may cause wasting of the muscles related to the joint. This occurs in two ways: (1) The inflammation may involve nerve-trunks passing the joint, set up a neuritis and produce motor, sensory, and trophic disturbances in the distal distribution of the affected nerves below the diseased joint. The process is simply a neuritis. (2) The articular filaments may be disturbed by the arthritis. Irritation ascends to the spinal centers and disturbs the trophic control of those cells related to the muscles physiologically associated with the joint and located on the proximal side of the affected articulation. It is a process acting through the reflex arc. We are to consider the second variety only. It is placed in this connection because it is a disease marked by disturbance of the spinal gray matter.

**Etiology.**—Any joint-lesion involving the articular filaments is competent to set up an arthritic muscular atrophy. It may thus follow simple, acute, or chronic arthritis, infectious arthritis, the arthritis of gonorrhea, of rheumatism, or of trauma. It has no proportionate relation to the severity of the joint-disease, but rather appears to be relative to the irritant qualities of the articular mischief.

**Symptoms.**—In acute joint-lesions the muscular atrophy usually begins within a month, sometimes within a week, and the muscles promptly show diminished bulk and altered contours. In chronic

<sup>1</sup> "Brain," 1889, p. 202.

<sup>2</sup> "American Journal of Med. Sci.," Oct., 1896.



arthritis—for instance, in gout and chronic rheumatism—it may very gradually develop, appearing very insidiously and requiring many months for its complete evolution. The striking peculiarity of the atrophy is that it is usually, and at first always, confined to the *extensors* of the joint, and involves them in their entire length. The only exception is in arthritis of the ankle-joint, in which case the calf-muscles waste. Arthritis of the shoulder affects the deltoid, of the elbow the triceps, of the wrist the extensors, of the fingers the interossei, of the hip the glutei, of the knee the anterior crural muscles, of the ankle the calf-muscles, of the toes the interossei of the foot. When the flexors are also involved, they suffer to a less degree. Sensory disturbances are absent, or are such as are referable to the arthritis. In occasional instances the joint-inflammation also affects the nerve-trunks in its neighborhood, and a neuritis is added, with symptoms below the joints. The wasting involves the affected muscles uniformly from end to end, and there is a



Fig. 128.—Severe atrophy of muscles in a case of long-standing arthritis.

corresponding loss of power. The electrical excitability of the muscles may be reduced slightly, but is generally increased relatively to the bulk of muscle present. The reaction of degeneration is never found. The tendon reflexes are exalted. In very pronounced cases a rectus-clonus or an ankle-clonus may be obtained. When the joint-disease subsides, the tendency is for the muscles to slowly recover, but in quite a proportion of cases they never completely regain their former vigor, and sometimes they remain permanently wasted.

**Morbid Anatomy.**—The atrophic muscles are flaccid, pale, and indurated. The fibers are simply diminished in size and there may be a little interstitial fibrosis. The only change in the nervous apparatus thus far recorded is in the nerve-terminals within the inflamed joint, due to their implication by the local disease. Better technique may be expected to discover cellular end-changes.

**Pathology.**—Paget, J. K. Mitchell, Volkmann, and Charcot early recognized the reflex mechanism of articular muscular atrophy. Volkmann found that arthritis experimentally produced was followed by the limited atrophy in question, and Raymond found that this atrophy did not follow if the corresponding posterior nerve-roots were divided. Hoffa clinised the matter by irritating joints on both sides and cutting the nerves on one side. The atrophy only occurred on the side retaining an unbroken reflex arc. The anatomical rule is that nerves supplying the extensors of a joint also innervate the joint. The

physiological association of arthritis and atrophy limited to the joint-extensors would in itself indicate the spinal element in the pathological mechanism. An interesting question is whether the symmetrical joint-changes and osseal conditions of gout and chronic rheumatism are not primarily due to the action of toxic substances upon the spinal cells governing the nutrition of bones and joints. Some cases of periarthritis or multiple arthritis symmetrically distributed, as well as the distortions of arthritis deformans and gout, strongly suggest a central disturbance as the principal and initial factor.

The diagnosis of uncomplicated arthritic muscular atrophy is usually easy. It depends: (1) On the limitation or excess of atrophy in the extensors of a joint actively diseased or formerly arthritic; (2) upon the quantitative electrical changes and absence of the reaction of degeneration, and (3) upon the increase of myotatic irritability. A microscopic section of the muscle would show a simple diminution of the muscle-fibers. When the joint-inflammation involves adjoining nerve-trunks, a neuritis may at the same time give symptoms below the affected joint. The atrophy usually remains sharply limited, in strong contrast to the invading tendencies of progressive forms.

The prognosis depends upon the joint-condition. If the arthritis subsides, the atrophy usually disappears. This is the rule in acute arthritis with early recovery. In more persistent lesions the atrophy is of corresponding duration and less likely to disappear when the joint recovers.

The treatment is primarily of the joint and secondarily of the muscles, by massage, electricity, and gentle exercises. Systemic arthritic conditions, of course, must be combated.

#### CHAPTER IV.

### LESIONS PRINCIPALLY AFFECTING THE WHITE MATTER OF THE CORD.

THERE are a number of diseases of the spinal cord in which the lesions are principally confined to certain white tracts, and they are frequently classed among the so-called system diseases or diseases of systematized lesions. The conception on which such a classification was based has been so much modified that it seems better to drop it altogether, especially as there has always been a great divergence of opinion as to what should be embraced under that caption. Acute poliomyelitis, for example, which was one of the usually accepted types, is, as we have seen, the result of accidental infection by way of the vascular supply. Posterolateral sclerosis, or ataxic paraplegia, is also usually based upon anatomical vascular conditions, and presents so many variations that it can not be considered as systematized from any point of view. In locomotor ataxia the spinal lesion is as sharply delimited as in any other, but it is a disputed question whether or not the cord-lesion is secondary



to initial root-changes. The morbid anatomy of the disease is as widespread that to class it as a systemic cord-lesion is to seriously contract the full view of the disease, and to ignore its neuritic and cerebral features. The secondary degenerations of the cord-tracts have been considered in connection with the various cerebral and spinal lesions which give rise to them. Primary spastic paraplegia, or Little's disease, has been discussed with the Cerebral Palsies (see p. 246), as it is dependent upon a congenitally unfinished condition of the upper neurons, and furnishes one of the diplegias of childhood.

Because the spinal symptoms of locomotor ataxia are the most salient of its many manifestations, and as its differential diagnosis is a constantly recurring problem in cord diseases, it is taken up at this time. In fact, however, it is a disease of the entire nervous system and must properly be so classed. The student should never lose sight of the fact that the cord-lesion in *tubes dorsalis* is only a small portion of its morbid anatomy.

#### TABES DORSALIS: PROGRESSIVE LOCOMOTOR ATAXIA.

*Tubes dorsalis*, *progressive locomotor ataxia*, *leucosyphilis posterior*, *atrophia of the posterior columns*, are some of the many names applied to a symptom-group of vast proportions. It is a disease always preceded by a syphilitic infection, usually marked by a degenerative process in the sensory nerves, posterior nerve-roots, posterior columns of the spinal cord, and often by similar changes in the medulla and cerebrum. Clinically, there is commonly disturbance of cutaneous and joint sensation; impairment of the muscular sense; incoordination of muscular movements, especially in the lower extremities; diminished or abolished knee reflexes and lessened pupillary reflexes to light; visceral and trophic disturbances and paroxysmal attacks of pain. The disease presents a progressive tendency toward complete helplessness.

In the description of this protran disease the masterly lectures of Marie<sup>1</sup> have been found of great help, and in some measure his arrangement of the subject is followed herein.

**Etiology.**—The principal factor in the etiology of *tubes dorsalis* is syphilis. After attention was first called to the postsyphilitic nature of locomotor ataxia by Fournier, in 1873, statistics on the subject showed an increasing proportion of cases presenting syphilitic antecedents. In 1894<sup>2</sup> he stated that in the examination of 720 cases he found the percentage of syphilitics varying from 87 to 93 for each hundred, taken separately. Erb reported 89 per cent., Déjérine, 92 to 94 per cent., Sachs over 90 per cent., Peterson, 71 per cent. Reports of cases of *tubes* acquiring syphilis subsequent to the development of ataxia cannot be obtained. Such immunity argues prior infection. Based upon the Wassermann test for syphilis, the Nogurli, Ross-Jones, and Lange tests of the spinal fluid and its cellular contents, one may now state with much more positiveness than is generally allowed in medical matters that without syphilis there can be no *tubes dorsalis*. Since Nogurli found the spirochete in the brain in 48

<sup>1</sup> "Leçons sur les Maladies de la Moëlle," Paris, 1892.

<sup>2</sup> "Les affections parasyphilitiques," Paris, 1894.

out of 200 cases of general paresis and in the spinal cord in 1 case out of 12 of tabes,<sup>1</sup> we may even go farther and say that there is no progressive tabes without spirochetal activity.

The tabetic symptoms may appear in from one to thirty-five years after the initial syphilitic sore, but ordinarily develop from the fifth to the fifteenth year. It is a noticeable and most important fact that the apparently mild cases of syphilis—those in which the secondary features are indistinct or entirely lacking—loom largely in the histories of tabetic patients. These are precisely the cases in which an intense medication is not urged or in which faithful pursuit of it by the patient was not secured. It must be said, however, that cases of tabes are only too frequently encountered in which syphilis has run a severe course and in which persistent and intensive medication has been heroically prescribed and faithfully borne for two and three years, and yet tabes has subsequently developed and after the usual interval.

It is rare for tabes to develop before the age of twenty-five years, just as it is rare for syphilis to be acquired before adolescence. In all cases appearing in childhood and youth, hereditary or acquired syphilis is present. The great proportion of cases appear between thirty and forty-five. As a rule, the alleged etiological factors of tabes, other than syphilis, are the very ones which contribute to the frequency of syphilis. Thus, the male sex is about ten times as frequently affected with tabes as the female. The same proportion obtains for syphilis. But among females subject to tabes Erb found the percentage of syphilis to be 80.5 per cent., practically the same as in males, and serological tests give the same results in both sexes.

The race question tells the same story. In rural communities and among the orthodox Jews cases of syphilis are comparatively rare, and tabes equally infrequent. Excessive worry has been accused of producing tabes. Its relation, if it has any, is by the increased exposure to luetic infection it entails. The action of cold, rheumatism, overexertion, alcoholism, and waste fevers has been much insisted upon in former years, but we know nothing definite about them. Many cases attributed to trauma have been misinterpreted cases of organic indiscriminate or combined cord lesions. In other instances the accident leading to the injury has been the result of tabes, not its cause, as in falls and fractures, the result of the previously unrecognised incoordination. Schittenhelm,<sup>2</sup> after reviewing the subject and the literature, reaches the conclusion that trauma as a unique cause of tabes is not proved, but that it may aggravate the tabetic condition.

A *neuropathic heredity* appears to play a predisposing part to some extent. We thus not infrequently encounter epilepsy, hysteria, chorea, insanity, hemiplegia, and diabetes in the ascendants of tabetics, and even, though most rarely, other cases of locomotor ataxia. May it not be that such instances indicate an inherited vulnerability on the part of a certain portion of the nervous apparatus to the toxic effects of luetic infection? It must be evident that only a very small percentage of syphilitics develop tabes. There is some ground for supposing that the spirochete may present a variable virulence which, under circumstances

<sup>1</sup> "Munch. med. Week.," April 8, 1912.

<sup>2</sup> *Ibid.*, 1903.



not now understood, may lead to the late lesions of tabes and paresis. There is an increasing amount of data tending to support the idea that there is a true *lues nervosa*. Head and Feinsilber<sup>2</sup> suggest a sensitization of nervous tissues by syphilitic activities making such parts particularly susceptible to the action of spirochetal remnants with consequent degeneration. In view of the almost invariable changes in the spinal fluid in early syphilis and the comparatively rare development of tabes the question is still an open one.

The bearing of occupation is that related to syphilis. Artists, actors, journalists, and soldiers are frequently affected, physicians and surgeons not infrequently, clergymen most rarely. Country laborers, so greatly exposed to traumas and exposure, very seldom present tabes or syphilis.

In spite of the immense importance of syphilis in the causation of tabes dorsalis, it must be kept in mind that the nerve-lesion is not comparable to the tertiary or secondary specific lesions, and is not similarly amenable to antiluetic remedies. It is a degeneration showing progressive sclerotic changes that are beyond repair. Following the views of Strumpell and Marie, we may hypothetically attribute the ascending degenerations in the nerves, roots, spinal cord, and brain to the action of a syphilitic toxin which primarily affects the cell-bodies of the fibers making up the afferent tracts. The progressive features of tabes may perhaps be correlated with the practically proved continuous activity of spirochetal life in these cases, and thereby the maintenance of a constant toxic factor.

Edinger undertakes to explain the apparently selective activity of the toxic agent upon certain physiological nervous tracts and structures by an ingenious hypothesis. Those nerve-elements which are the most constantly employed are most affected. Thus, the sensory tracts, especially in the lower parts of the cord, the papillary activities, the bladder function, and the intestinal field are early and commonly affected. The hypothesis confines the elements of (1) fatigue locally precipitating the effects of (2) a general toxic state.

The theories as to the pathogenesis of tabes are numerous and varied. Four principal ones may be mentioned. First, the toxic theory with syphilis as the usual origin of the toxin. Second, the strangulation theory of Obersteiner and others who find the initial pathological factor in pial thickening, whence arise the posterior root and cord changes. Third, Marie's recent contention that the changes start in a lymphangitis of the posterior columns of the cord; and, fourth, the "Ersatz" theory of Edinger above indicated. Orr and Rowe,<sup>3</sup> while granting a hereditary or acquired weakness of the nervous apparatus, believe that the process is located in the sensory neurons of the posterior cord tracts, because of the anatomical absence of the neurilemma at the point where the sensory root fibers enter the cord. They find this to be the initial point for the degenerative process. Nagotte has insisted upon the importance of a posterior meningitis to which he considers that the posterior root changes are secondary.

**Morbid Anatomy.**—In this section it is purposed to take up the morbid anatomy of the nervous apparatus in tabes dorsalis. The morbid

<sup>2</sup>"Brain," 1914, p. 1.

<sup>3</sup>Ibid., Winter, 1904.

anatomy of the trophic lesions in the bones, joints, and soft structures will be described respectively with their clinical features. As the disease is essentially marked by ascending degenerations, it is well to commence at the periphery.

The *spinal nerves* are usually found to present a degeneration which is greatest at the periphery and most marked in, if not entirely confined to, the sensory fibers for the skin, muscles, and joints. The trophic centers for these fibers are in the posterior root-ganglion in part, but it seems probable that there are also superficial peripheral trophic cells



Fig. 180.—Sections of the cord of an early stage of tabes dorsalis. 1, Lateral region; 2, dorsal region; 3, ventral region (Meyer).

for at least a few of them. The *muscle spindles* which undoubtedly have a sensory function are constantly found degenerated. By some writers these nerve-changes are designated a *neuritis*, but histologically the changes conform to a Wallerian degeneration. The *spinal nerves* consist

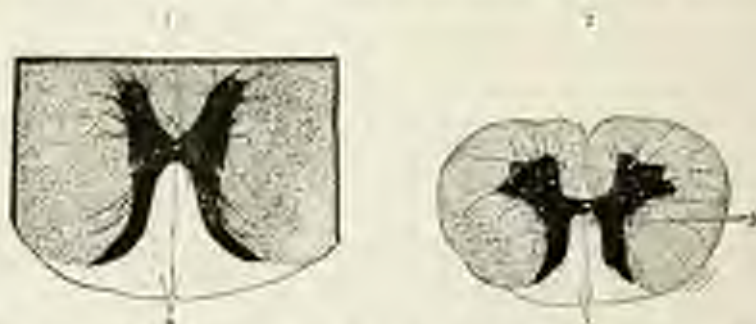


Fig. 181.—Sections of cords in advanced tabes. 1, Lateral region; 2, ventral region. Arrows show by small region (Meyer).

present a marked difference between the anterior or motor members, which are ordinarily normal, and the posterior sensory roots, which are usually, if not invariably, greatly degenerated. Only when changes have taken place in the anterior cornual cells do we find changes in the



anterior roots, and then there is corresponding amyotrophy. This is usually a late and secondary or accidental feature in tabes. The posterior roots and their ganglia show much disease. Oppenheim, Sommerling,



Fig. 181.—Section in lower dorsal cord, in a case of advanced tabes showing complete atrophy of the posterior columns and of Clarke's cellular columns (Marec).

Marie, Obersteiner,<sup>1</sup> Juliusberger and Meyer,<sup>2</sup> describe changes in the root-ganglion cells, which are found reduced in size and number. Nuclear displacement and granular changes were demonstrated by Nissl's method, according to the last-mentioned authors. The fibers within the ganglia were also atrophic. The posterior roots are practically always degenerated. The posterior root-ganglion occupies, therefore, a prominent position in the development of tabetic lesions, as it exercises a trophic control, both downward over the sensory

fibers of the nerves and upward over the posterior root-fibers and their continuation within the posterior tracts of the cord. Changes in the visceral branches of the sympathetic nerves of a degenerative character have also been reported, and degeneration of the fine fibers of the sympathetic arising from the viscera and entering the cord by the posterior roots has been demonstrated in a series of cases.<sup>3</sup>

To understand the distribution of the cord-lesions it is necessary to recall that the posterior root furnishes three sets of fibers, which enter the posterior horn by different routes and at different levels. The fibers of the first group, almost as soon as they reach the cord, enter the posterior gray to the inner side of the horn in its posterior third by a short route through the tract of Lissauer. Those of the second group pass to the inner side of the first in Burdach's column, and enter the middle third of the horn's inner border at a level considerably above their point of entrance to the cord. Some of them reach Clarke's column. The third group, arising mainly from the lower limbs, enter still nearer the median line and pass up nearly the entire length of the

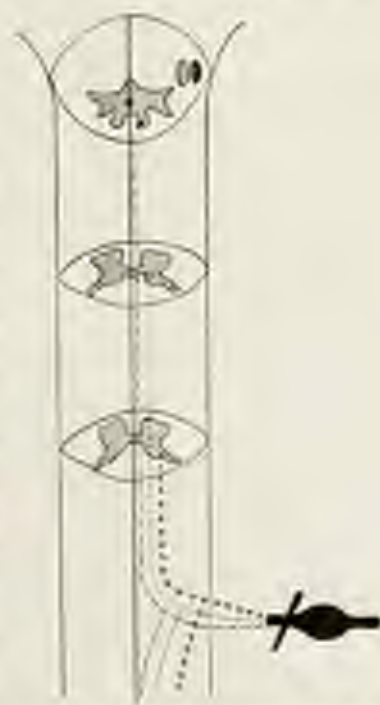


Fig. 182.—Schematic representation of the routes provided by the sensory root-ganglia, entering the cord from the root-ganglia (Marec).

<sup>1</sup> Berlin Klin. Wochenschr., Oct. 14, 1897. <sup>2</sup> Neurolog. Centralbl., Feb. 15, 1898.

<sup>3</sup> Bence, "Lesions of the Sympathetic System in Tabes," Paris, 1900.

cord, constituting the columns of Goll, and terminate in the bullar gray. The tract of Lissauer lying between the head of the posterior horn and the periphery of the cord among the entering posterior-root fibers, is made up of fine fibers arising from the posterior roots either as collaterals or as direct continuations of the numerous finer fibers of the root. These fibers of Lissauer's tract, after ascending a short distance, enter the gelatinous substance of Rolando, which caps the posterior horn, and some of them penetrate the posterior horn itself. This tract also degenerates in tabes.

Embryologically, the posterior columns of the cord, including Lissauer's tracts, are an accession to the cord. They arise in the lateral neural plaques, which are finally represented by the posterior-root ganglia, and enter the cord by way of the posterior roots, pursuing their course upward in the manner indicated. This part of the cord only reaches full development at birth. It is to this posterior, exogenous, independently developed portion of the cord-structure that the spinal lesions of tabes are principally confined.

These vary at different levels and in different stages of the disease. In long-standing cases the entire portion of the cord enclosed between the posterior horns and the commissure may be sclerosed from the filum terminale to the medulla. In incipient cases the tracts of Lissauer and the columns of Burdach are affected at the lowest level, the columns of Goll above. Ordinarily, the sclerotic changes of tabes are most marked in the lumbar cord, where they commonly begin; in other cases the cervical enlargement is most affected; again, in others, the intramedullary lesions of the medulla predominate, and when associated with cortical changes we have the progressive paralysis of the insane. Clinical varieties correspond to these various preponderating locations of the sclerotic process, and all intermediate varieties and combinations of lesions and clinical types are encountered. This sclerotic change, like that in the nerves, is a pure Wallerian degeneration.

The lesions of the spinal gray in tabes, as far as now describable, consist of a degeneration of the fine nervous reticulum about the cells of Clarke's column near the inner border of the neck of the posterior horns. The change is an early one and most marked in the outer cells of the group. This reticulum is made up of the terminal brushes of fibers from the posterior roots. Ordinarily, the cells themselves are spared, but in some cases they, too, show degenerative changes, and the direct *cerebellar tracts* and *Gower's ascending antrolateral tracts* are then also sclerotic. These two cord-tracts find their trophic supply in the cells of Clarke's column; hence their degeneration when these cells are affected. Clarke's column begins in the upper lumbar segments and extends to the seventh or eighth dorsal, when it becomes extremely attenuated and practically disappears at the second dorsal, to again appear above the cervical enlargement in the upper cervical cord. The lesion of Clarke's column is, therefore, most marked in the lower dorsal region.

The posterior horn proper, according to Lissauer and others, shows only insignificant changes except in the caput of Rolando, where the fine fibers and radiating fibers from the posterior roots are customarily degenerated.



The cord-lesions in tabes are commonly symmetrical, but not infrequently in early stages one side will show more sclerosis than the other, often corresponding to greater symptoms in the limb or limbs of that side.

In addition, it is to be noted that there is a *clouding of the meninges* overlying the sclerotic tracts in the spinal cord, to which much importance has been given by some who see in it a *meningo* forming an initial pathological process in tabes. This and the cord-changes can be frequently recognized by the eye, but in incipient cases recourse to the microscope is required to decipher the morbid condition. In some instances the meningeal condition reaches one of chronic inflammation, which, in turn, may cause a *marginal syphilis* that may even extend to the pyramidal tracts.

Bearing upon the question of meningitis is the fact that the *spinal fluid* during life commonly presents a lymphocytosis, as has been abundantly proved by Vidal, Sicard, Ravaut, Schenbrun, and many others. From 10 to 20 cells in the cubic millimeter of freshly drawn spinal fluid is considered by Nonne as a weak positive finding; from 20 to 60 positive, and above 60 strongly positive. In the great majority of cases, over 90 per cent., there is an increase of albumin in the form of globulin in the spinal fluid as determined by the tests of Noguchi and Ross-Jones, the Phase I of Nonne. At the same time the Wassermann serum reaction is positive in 70 per cent. of all cases of tabes both in the blood and spinal fluid. The colloidal gold test of Lange also gives a definite positive result in at least 80 per cent.

The *cranial nerves* are all liable to degeneration, but this tendency is most marked in the optic and auditory nerves, which in effect are cerebral lobes. Changes of a similar sort are not rarely found in both roots of the trifacial or in the nuclei of the motor nerves of the eyeball, and of the intra-ocular muscles, in the glossopharyngeal, pneumogastric, facial, and hypoglossal. Involvement of the iridociliary apparatus is, perhaps, the most common of all.

The *cerebral lesions* of tabes, aside from those of the cranial-nerve nuclei or even embracing them, are those of *parietic dementia*, the description of which is contained in the second half of this volume. With this disease tabes has the most intimate relations. Fournier has gone to the point of considering them of identical nature and only varying in the primary location of the lesions. The syphilitic theory of parietic dementia rests on exactly the same sort of a foundation as does that of tabes dorsalis. A certain proportion of cases of tabes develop paralytic dementia; a large number of parietic dementics present tabetic symptoms and tabetic cord-lesions. Both diseases commonly have identical bulbar and cranial-nerve symptoms and lesions. Though Jendrassik tends to consider the cerebral lesions of tabes as always initial, they are probably concomitant only, and due to the same specific cause. Their association in point of time and development is open to all possible modifications throughout all the varieties of each affection. The most pronounced types of both syndromes are presented as the sclerotic process falls first and most severely, respectively, on the caudal or cephalic extremity of the cerebrospinal system.

**Symptoms.**—From the extent of the morbid anatomical changes in tabes it must be evident that its symptomatology embraces nearly every bodily organ and function. The clinical features of the disease can only be fully presented by taking them up systematically. Afterward an attempt will be made to group them in describing the common clinical varieties of the malady. As we proceed, the relation of the various symptoms to the course of the disease will be indicated. Their relative frequency will be shown in tabular form.

**Motor Disturbances.**—The motor disturbances of tabes, while not the earliest, are among the most important, and are the ones which usually first seriously attract the patient's attention. They consist of: (1) *ataxia*; (2) *incoördinary movements*, and (3) *pulsio*.

Tabetics deprived of their muscular sense by the posterior sclerosis or by the degeneration of the peripheral sensory fibers in the muscles and joints can not determine the exact positions of their limbs without the aid of vision. This is, no doubt, increased to some extent if attended by cutaneous dysesthesia, which is frequently present. They thus "lose their legs in the bed" in marked cases, and have to verify the position of their limbs with their hands or eyes. In less pronounced cases they can not with closed eyes duplicate with one limb the position passively given the other by the examiner, or do so with uncertainty. Another manifestation of the perturbation of the muscular sense is an inability to accurately distinguish the difference in weight of similarly shaped objects held in the hands. The normal individual can usually determine a difference of five per cent. This symptom can sometimes be shown by having the patient first write a short sentence with open, and then

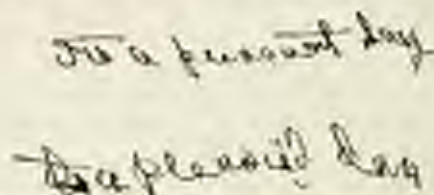


FIG. 184.—Ataxia of the upper extremities shown in the handwriting. 1, written with eyes open; 2, with closed eyes.

repeat it with closed eyes. The uncertainty of position and the consequent incoördination is then sometimes graphically demonstrated, while grosser movements with the upper extremities may be tolerably exact. Many patients fail to touch the nose or ear or any given point with the index-finger when the eyes are closed.

Tabetics commonly present the sign of *Romberg* early if the lower portion of the cord is involved. With closed eyes they sway or may fall heavily if the feet are placed close together, and may even be unable to stand with a broad base of support. Walking backward with closed eyes will almost invariably determine incoördination in the lower extremities if present in the slightest degree. So will attempts to stand on one foot. In advanced cases this loss of muscular sense causes so marked an ataxia that walking is impossible, even with the eyes open.

To this source we must also attribute the *obscuro gait* that is so characteristic when well developed. It by no means appears clearly in all



cases, but may usually be induced to some degree by having the patient walk with closed eyes, or may be manifest in the difficulty of executing a prompt "about face," in suddenly rising from a chair and attempting to immoediately walk forward, in descending a stair, in halting suddenly when advancing, and in marking time after the military fashion. In crossing one knee over the other the moving limb is often raised too high and too vigorously dropped across its fellow. When the gait is plainly staccé, the patient rises from the chair with some suddenness, as if lifted by a spring, then balances a moment before putting himself in motion. The first steps are usually taken briskly and the patient may even plunge forward. The legs are thrown out widely. The foot goes forward irregularly with some rigidity, the toe up, often departing laterally from the direct line, and is brought down with a shock upon the heel, or flat-footed. As the body is advanced, there is a tendency to overextension of the supporting knee-joint, which often produces a backward leaning of the limb and may constitute a marked deformity.



FIG. 184.—(Left) is taken. Shows exaggerated extension of supporting knee, (right) of advancing leg, (dotted line, knee). According first, articulation of knee, and assisted by cane.



FIG. 185.—Showing the tendency to over-extension of the knee-joint, in cases of severe disease of the cord.

The foot is usually raised too high at the end of the step, but is also prone to catch upon any trifling obstacle as it swings forward. The opposite foot is then advanced in a similar irregular way, and the patient almost invariably aids himself by carefully watching his feet and by the additional assistance of one or two canes (Fig. 185). In some cases there is considerable uniformity in the manner of taking each step, but as often no two paces are exactly alike, and the patient, instead of advancing in a straight line, swerves more or less to one and the other side, often bumping into neighboring objects.

When he sits down he does so with suddenness, as if both knees gave way too soon, as they often do. In the same way he can not gradually lower himself from the erect position to a squatting attitude, but drops suddenly when the knees are partially bent. The sudden giving way of a knee sometimes causes him to fall, and, as a rule, his attitude and gait denote every effort to trust these joints as little as possible and to fix them by overextension.

When the upper extremities are markedly ataxic, it is shown in the manner of grasping a small article, toward which the hand goes with wide-spread fingers, and grabs down on it as if it might escape by flight. Finally, walking becomes impossible, and the upper extremities may become useless except for wide and inexact movements. Commonly inco-ordination is most marked in the lower extremities, but in the cervical type of tabes the condition may be more pronounced in the arms. In all these motor affections, however, muscular power is not necessarily impaired. It is only the muscular sense that fails. The ataxia is commonly of insidious development, but in some instances comes on abruptly, usually as the result of some physical strain or exhausting condition or after a period of disease of the limbs, as, for instance, after an illness or fracture. In cases where it is slowly increasing it may show sudden intensification, some of which may recede, but rarely does such sudden increase of ataxia fully pass away.

*Jarshberg* movements in tabes dorsalis are not uncommon, and are probably more frequent than reports indicate, as they are obscured by the major features due to inco-ordination. In the early as in the late stages they may be observed in some cases, and usually are uniform for the given patient. They may consist of a sudden involuntary movement of the thumb or a finger, or the turning of the wrist, or the jerking of the arm or foot. In a case now under observation the entire lower extremity is frequently violently jerked, mainly by the flexors of the hip, and these movements may be repeated rapidly, but irregularly, for several minutes, and even for an hour or two daily, irrespective of fatigue or position. Such jerkings of the legs are prone to occur during sleep. In exceptional cases and in their terminal stages both lower extremities may be forcibly drawn up to the body. This occurs upon coughing, sneezing, or sometimes upon voluntary efforts of any sort or even upon the manipulation of the extremities. The slighter movements have been called *athetoid* by some, but they present nothing in common with the true *athetoid*, vermicular movements so common in the cerebral palsies of children.

Frenkel has recently emphasized the fact that *argyria* is easily provoked, in the muscles which show diminished reflexes, by sharply pinching them, or by a smart light blow with a ruler, at right angles to their length.

*Palsies.*—The paralytic features in tabes are of two orders, and are to be clearly distinguished from the loss of coordinate power which constitutes the major motor difficulty. (1) One is due to the wasting of the muscles, a result of the involvement of the anterior horns of the cord, usually appearing late in the disease; (2) the second group, to which attention is here confined, is the result of organic or vascular changes



in the cerebrum or cord. They appear in about one-fifth of the cases, and embrace hemiplegia, facial paralysis, lingual paresis, monoplegias, laryngeal palsy, and paraplegias, the last being due to focal disturbance in the cord. In addition, paralytic drooping of the lid constituting ptosis, one of the early manifestations of tabes, may be mentioned, but will be more fully described under disturbances of the ocular apparatus. The facial palsy also may be neuritic.

A peculiarity of these palsies, as pointed out by Fournier, who has tabulated them, is their usual benign and fleeting character. They may last some days or a few weeks, when they may completely and spontaneously disappear, but a minority of the palsies remain permanently. Some of these transitory cases undoubtedly are to be referred to hysteria, which not infrequently is a complication of tabes. The permanent cases are probably due to vascular or inflammatory accidents in the brain or cord; in other instances a neuritis may determine a localized loss of power.

**Sensory Disturbances.**—The sensory disturbances in locomotor ataxia are among the very earliest to appear, and persist in some form or other throughout the course of the disease. They are rarely the same in any two patients, and may vary almost infinitely in a given case.

**Subjective Sensory Disturbances.**—The first group of sensory disturbances are those of a subjective nature, regarding which we have to depend entirely upon the descriptions of the patient. They may be subdivided into those which are intermittent and those which are permanent. First and most important of the intermittent variety are the so-called *lightning pains*, which are experienced in the face, extremities, or trunk, but most commonly in the legs. They are described in the most vigorous language and the most striking terms by these unfortunate sufferers, are clearly atrocious in character, and are not associated usually with any evidence of disturbance in the part where they locate, but exceptionally after severe pains ecchymosis or some edema may be found.

Pains of a similar character, but somewhat less in intensity, and transitory like the lightning pains, are *lancinating*, *burning*, *burning*, *twisting*, *venous* of a painful character. In each patient an attack of such pain is likely to be followed by a similar attack in a similar location, and these attacks or crises occur in some cases with a degree of regularity every week, month, or year. Ordinarily, they are attributed to rheumatism, and it is not uncommon for patients to be treated for rheumatic disturbance for months and even years before the tabetic nature of the painful affection is recognized. A careful examination in a case marked by repeated painful attacks will almost invariably determine other evidences of tabes. The diagnosis should be made. When commencing early in the disease, the pains may disappear after the ataxia has become prominent, but if they do not appear in the early stage they are not likely to appear later. In some cases they persist through the entire duration of the disease. Cases presenting very severe pains early seem to run a more tardy course than those in which pain symptoms are insignificant.

Another variety of subjective pains are associated with the viscera,

and constitute nasal, testicular, ovarian, urethral, vesical, gastric, laryngeal, and intestinal crises, which will be taken up in the consideration of the visceral disturbances of the disease.

Tubercles occasionally complain of attacks of muscular cramp, which, like the lightning pains, have a tendency to come on in gusts, and frequently compel the patient to take to his bed. Their common location is in the muscular masses of the lower extremities or in the small of the back.

The second class of pains is more permanent in character, lasting weeks, months, or even years, perhaps appearing and disappearing several times during the course of the disease. One of the most important of these is the *girdle sensation*. The patient describes it as the feeling of a tight belt, and may frequently attempt to relieve it by loosening his clothing. It may be situated in the lower portion of the abdomen, or at the level of the umbilicus, or about the chest. It is usually rather narrow in its vertical extent, but may be of considerable width, giving rise to sensations of an iron or rigid corset. An analogous sensation is sometimes felt in the extremities as of a bracelet, or as if the legs or arms were bound with rope. Paresthetic sensations of a more or less persistent character are described by the patient as the creeping of ants or insects, a feeling of fullness in certain parts of the body, especially along the ulnar border of the forearm and hand, and in the lower extremities below the knees. In other cases the sensation is that of a cobweb or some light fabric upon the skin.

**Sensory Disturbances Partially Objective.**—*Analgesia* is one of the commonest manifestations of tabes, affecting not only the cutaneous extent, but the muscles, bones, and joints. Even dislocations and fractures, perforating ulcers, and other diseases of the deeper parts, as well as of the skin, are frequently unattended by pain. Very often the patient is unaware of the analgesia, and is surprised to observe that a needle can be thrust deeply into the tissues without giving rise to any discomfort. This loss of the sensation of pain is frequently distributed in plaques upon the trunk and extremities without reference to the ordinary manifestations of the disease, and does not spare the surface of the



Fig. 107.—Tabes dorsalis. (Front and back views.)

head. It has a tendency to symmetry, affecting both upper extremities, both lower extremities, or the trunk bilaterally. On the head, however,



there is a tendency to a unilateral distribution of the analgesia. On the trunk the most common location for the analgesia is over both pectoral regions, about the umbilicus, in each inguinal region, and over the shoulders. Frequently these analgesic plaques are marked by a hyperæsthetic border. On the upper extremities the analgesia most commonly affects the fingers or the ulnar border of the forearm. In the lower extremities it is the sole of the foot, the heel, and the toes; on the thighs the inner surface, corresponding to the adductors. The nerve-trunks, as the ulnar at the elbow and the external popliteal at the head of the fibula, are frequently found insensitive in very early stages of the disease. The testicle commonly loses its normal sensitiveness to pressure.

On the trunk is frequently found what may be denominated the *tubercle cutané*. In about four-fifths of the patients, even at an early stage, there appears a band about the chest, which may be most marked on the anterior or more commonly the posterior surface, or rarely confined to the pectoral regions. This area presents a diminished sensation to touch (Lager, Patrick, Boiss). Its outline above and below is sometimes hyperæsthetic. It varies in vertical width from three or four inches to an area which would be covered by a corset, and is sometimes associated with a girdling sensation. This cutaneous distribution is not ordinarily one of analgesia, but of tactile loss, though it may be both. Its limits are those of the cutaneous extent of the spinal segments, and do not conform to the course of the intercostal nerves. These areas of tactile anesthesia have four principal localizations: (1) In horizontal patches or girdles on the trunk; (2) the internal surface of arms and forearms and ulnar borders of hands; (3) the perineal and genital regions; (4) the outer margins of the feet, outer sides of legs, and the inter-internal surfaces of the thighs. They are often related to the painful disturbances of neighboring viscera.

The sensibility of the bones is reduced. This is shown by the reduction or loss of perception of the vibrations of an active tuning-fork held in contact with subcutaneous bony parts. Rarely occurs painless joint operations done without anesthetics. Probably all the deeper parts share in the analgesia.

*Hyperalgesia* is a common condition in tabes. It may appear in plaques similar to those of analgesia, but has less tendency to symmetry of distribution and is less persistent. These hyperalgesic plaques frequently are the seat of lightning pains and often appear during the painful crises. Hyperalgesia may be found not alone for the painful stimulus of the needle, but for cold and other sources of pain. Many patients in the early years of the disease find hot water intolerable in the bath and frictions by hand or towel almost unbearable.

In some instances the stereognostic sense is materially impaired and in a ratio disproportionate to other sensory modifications.<sup>2</sup> Thus, in the hands, which may be the seat of only slight paresthesia, a match-box may not be told from a coin and other familiar objects similarly mistaken or unrecognized.

Modifications of the cutaneous sensations are quite frequent. Com-

<sup>1</sup> Marmoreau, "Sem. Méd.," Oct. 13, 1897. <sup>2</sup> Boiss, "De Méd. Jour.," Feb. 7, 1901.

namely the *retardation of sensation* from the extremities is referred to, so that the patient, when instructed to do so, does not indicate the perception of the pin-prick upon his shin or foot within a period of three, five, or even ten seconds, or more. In a general way the retardation of the transmission of sensation increases with the distance of the part from the head, not only because of the distance, but from the fact that the extremities usually present the greater disturbance of sensation, probably owing to the changes in the distal ramifications of the sensory nerves. A peculiarity is that the retardation of sensation may be dissociated. Painful sensation may be retarded, while that for touch is not, so that the patient feels the prick of a pin immediately as a touch and subsequently as a pain. Frequently patients are unable to distinguish the character of the stimulating impression, recognizing a prick as a pinch. This, in other words, is an expression of the diminution of their sensitiveness. Tabetics may fail to properly locate the stimulus,—a pinch on the foot may be referred to the knee or to the opposite foot. In general, the tactile sensations are abolished later and to a less degree than sensations of pain, but they are also frequently modified, and this gives rise to additional difficulty in locomotion. The patients express themselves as having a feeling of walking upon a thick carpet, upon cushions, upon rubber, or other yielding substances.

It may be found that a stimulus not at first recognized is apprehended upon being repeated a few times with some rapidity, the association of effects being competent to reach a sensorium cut off from a single impulse. Again, a stimulus at first competent may, upon repetition, fail to rouse the sensorium by exhaustion apparently of the conduction apparatus which, after a short interval of rest, again responds to the original excitation. Even the synergic dissociation of extensor sensibility has been encountered in tabes, but commonly it is devoid of these exact and equal boundaries for all forms of sense anomalies which are found in the true synergic syndrome.<sup>1</sup>

In all cases it is necessary to use the utmost caution in making tests of sensation, as already indicated in Part I. Some allowance also must be made for the intelligence and temperament of the given patient.

**Disturbance of the Reflexes.**—The *knee-jerk* is loosened, unequal, or more frequently abolished, and that at an early stage of tabes, in at least nineteen out of twenty cases. The lost knee-reflex, often called *Wiedehopf's sign*, must be sought with great care, but in no instance should the patellar reflex be considered extinct unless the plan of reinforcement and all precautions are taken to elicit it. It may also be well to recall that it is diminished in advanced age, in sleep, by fatigue, in exhausting illness, and by any condition, such as a peripheral neuritis, that destroys the afferent and efferent paths or the spinal center. It is also possible that very rarely a healthy adult may be found without a knee-jerk. The *Achilles reflex* ordinarily fails with the knee-jerk. In fact, it may disappear before the knee-jerk is lost and constitutes a valuable early test. The reflexes in the upper extremity fail when the cervical cord is involved, and Fraenkel claims that the triceps reaction is lost as commonly and as early as the knee-jerk;<sup>2</sup> but

<sup>1</sup> Raymond, "Léçons," Paris, 1900.

<sup>2</sup> "Deutsch. Zst. f. Nervenzh.," July, 1900.



this is clearly erroneous. The *superficial reflexes*, such as the plantar, abdominal, dorsal, and scapular, are variable. The *iris reflex to light* is



Fig. 100.—Abnormal range of motion of hip-joint due to hypotonia in sides.



Fig. 101.—Abnormal abduction of thighs, the "split" position due to hypotonia in sides.



Fig. 102.—Abnormal flexibility of spine and hips in lateral view in hypotonia.

usually abolished early, but will be considered later with the disturbances of certain other organic reflexes, including those of the cremasters.

All muscles in the involved areas present a peculiar *lack of tonicity*, of which the reduced or lost tendon reflexes are a manifestation. This

hypotone is readily demonstrated by the ease with which over-extension can be imparted to knees, ankles, and elbows, and in the great range of flexion at the hip and of abduction of the thighs.

**Disturbance of the Visual Apparatus.**—Both the external and internal portions of the ocular mechanism are frequently impaired in tabes.

*Prosa and squints*, usually unilateral, sometimes bilateral, are of common occurrence in the pre-taxic as well as in the later stages of locomotor ataxia. They may be and often are temporary and fleeting, almost momentary, but show a marked tendency to recur and occasionally are permanent. Careful questioning will commonly recall to a tabetic's mind some such ocular experience. Its temporary character is the best evidence of its tabetic, we may even say of its syphilitic, nature. Any of the extrinsic muscles of the eye may be selected by the disease, but those under the control of the third cranial nerve show more than their due proportion of paralytic disturbances. They may be gradually invaded, and a progressive external ophthalmoplegia results with permanent disability. *Loosening, exophthalmos, enophthalmos, nystagmus*, and reduced ocular tension on one or both sides, singly or in combination, have been noticed in rare instances.

The *pupils* are affected in the great majority of tabetic cases, and furnish some of the earliest and most important diagnostic signs. Every possible pupillary modification may be encountered in tabes,—*inequality, irregularity, salacia, anidriasis, sluggishness, loss of light reflex, loss of accommodation reflex, loss of reflex to pain, and absolute blindness*. There is only one other disease that has a parallel in this matter of

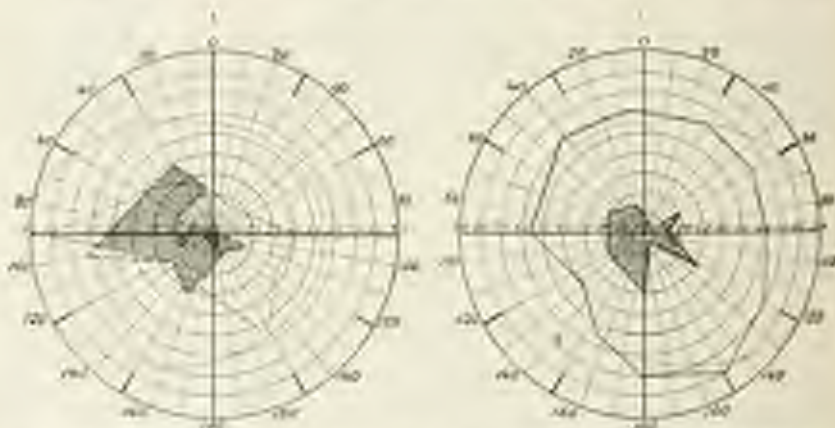


FIG. 10.—(Continued) irregular (cont.) fields in tabetic optic atrophy. 1, Left eye, showing the normal form field; broken line—smaller field (arrows) in which there is a slight anomaly, small central field in blue green; 2, right eye; irregular form fields (blue lines); small inner field in blue green.

pupillary disturbance, and that is parietic dementia. The analogy, if not identity, of these diseases has been sufficiently indicated. These various pupillary disorders may be combined in any and every way. Sluggishness of the pupils to light and slight inequalities and irregularities of outline are usually encountered very early in the disease. Later,



contracted pupils still responding to accommodative efforts but not to light, constituting the *Robertson pupillary sign*, are noted, and still later loss of reflex to pain, and eventually complete iridoplegia, with or without paralysis of accommodation, is often found. Dilated pupils may be subsequently contracted, but pupils once contracted to an extreme degree very rarely again dilate widely. The fixed pupils of tabes resist agents that ordinarily control the pupillary muscles, and if forced from their abnormal proportions, as by belladonna, return to them only after several weeks, and sometimes not at all. The dissociation of the light and accommodative reflex is, perhaps, the most important of all ocular symptoms, and appears early in over one-half of all cases. Over seventy per cent. of tabetics show some pupillary disturbance.

The optic nerve degenerates in about ten per cent. of tabetics, and this occurs in those patients who have shown ocular palsies more frequently than in others. The optic atrophy is usually bilateral, but has a tendency to attack the left eye first. Its natural termination is in blindness. In rare instances it comes to a standstill, or even recedes a trifle. It may cause blindness in a few months, or many years may be required for the extinction of sight. As a matter of fact, the optic atrophy may have advanced to a considerable development before the patient notices any visual impairment. The order of symptoms is usually: (1) A contraction of the color-fields, green, red, and blue fading in the order named; (2) the form-field shrinks, and (3) vision begins to diminish. The retraction of the field is usually most pronounced on the temporal side, but may be irregularly concentric. In occasional instances hemianopia or quadrant defects have been observed, and even central scotomata. These partial fields are, perhaps, properly attributed to a retrobulbar neuritis. The characteristic ophthalmoscopic picture from the first shows a blanching of the papilla, which becomes grayish or bluish-white and pearly. Its border is sharply defined, and sometimes it is stippled by the ciliiform markings. Eventually the vessels diminish in size, first the arteries, then the veins.

Optic atrophy may be a very early tabetic feature, and usually appears either before or during the early portion of the ataxic stage, while the patient is still walking fairly well. It is a clinical fact of much value that, as a rule, the cases developing optic atrophy early do not show much ataxia, and the locomotor difficulties are only those of blindness. When appearing in the ataxic stage there is little or no further increase of incoordination, and in certain cases the ataxia almost or quite disappears. Benedikt says it invariably subsides.

**Auditory Symptoms.**—Morgagnio<sup>3</sup> has shown that eighty per cent. of tabetics present auditory defects which may appear early or late in the disease.

The auditory nerve is subject to a degeneration similar to that noted in the optic and about as frequently. In other instances the disturbance is in the middle or external ear. The impairment of hearing is frequently insidious and extremely slow in reaching a complete degree, but in some cases comes on with rapidity or even abruptly. It is usually bilateral but commonly more marked on one side than upon the other. The dis-

<sup>3</sup> "Archiv. f. Otolaryng., 1890.

tion between disease of the nerve and disease of the conduction apparatus is determined by Rinne's test (see page 65). Weber's test, which consists of placing the handle of a vibrating tuning-fork on the vertex, enables the patient to thus hear the note better in cases of middle or external ear disease than when the nerve is affected. The disease of the middle ear is, perhaps, sometimes of a dystrophic sort.

Many tabetics are troubled with *flashes* and vague subjective auditory sounds. Others have constant or paroxysmal attacks of *moral vertigo* that may be sufficiently severe to provoke vomiting and great prostration. Auditory *hyperæsthesia* to electric currents was found by Marina<sup>1</sup> in eight out of eleven cases of tabes, which also indicates the frequency of disturbance of the auditory apparatus in locomotor ataxia. Bonnier<sup>2</sup> attributes to labyrinthine disturbance many of the common signs and symptoms of the disease. He enumerates deafness, vertigo, ataxia, nystagmus, diverse disturbances of ocnomotricity, pupillary changes, and a large number of others, and found labyrinthine irregularity in 80 per cent. of cases examined.

The senses of smell and of taste have also been found impaired in rare cases, even to the point of complete loss. A careful examination for similar defects in all cases would probably show them to be somewhat common. Klippel and Julian<sup>3</sup> have reported nasal crises marked by curious sensations in the nose and nasopharynx and violent bouts of sneezing.

**Visceral Disorders.**—The visceral features of tabes are among the most interesting and constant manifestations of the disease, and among these most frequently overlooked and misinterpreted by the physician. They consist, for the most part, of paroxysmal attacks attended by pain and disturbed function of some viscous, as the stomach, intestine, or bladder, and are called *visceral crises*. Other visceral disorders are of a continuous character, and are attributable to impairment of the nervous and vasomotor control of the parts. Though any viscous may be affected in tabes, the crisis features for the given patient are usually limited and uniform in their manifestations.

**The Stomach.**—*Gastric crises* in tabes are very common. They are characterized essentially by pains and vomiting. The pains are located in the pit of the stomach and often radiate in various directions. They are occasionally referred to the heart; indeed, angina pectoris may appear with them. They are intense, and sometimes so severe as to seem unbearable, and may actually cause insensibility. Sometimes they strike through to the back or flash into the flanks and through the abdomen. The vomiting is repeated and intractable. Sometimes it is attended with excessive straining, and again the gastric contents are ejected with very slight cruetative efforts. The vomitus at first consists of undigested food and then of gastric mucus in large amounts, and finally of bilious mucus in the protracted attacks. The vomiting is frequently repeated, only a small amount being ejected at a time after the first efforts. The slightest ingestion of food or liquid of any sort promptly provokes a repetition of the emesis. Sahli, Hoffmann, and

<sup>1</sup> Arch. L. Psych.,<sup>11</sup> t. xxi, p. 136.      <sup>2</sup> Nerv. Ton., de la Salpêtr.,<sup>11</sup> 1896.

<sup>3</sup> Rev. de Méd.,<sup>11</sup> Jul. 18, 1900.



others have shown that there is hyperacidity due to an increase of hydrochloric and lactic acids, most marked at the beginning of the attack and gradually diminishing as it proceeds.

The gastric crisis is usually attended by a state of marked prostration that may even recall the collapse of the algid stage of cholera, and is usually equal to that of severe seasickness. The patient, cold, blanched, and covered with profuse perspiration, presents the appearance of severe shock.

Gastric crises, like all the critical manifestations of tabes, are of sudden onset and abrupt termination. They may last an hour or two, or several days or weeks without intermission. In spite of the great thirst that attends prolonged attacks, the smallest amount of liquid is not tolerated by the stomach, and alimentation *per os* is out of the question. Suddenly the patient may feel hungry, the pains may abruptly cease, and both food and drink may be taken freely without further disturbance.

Gastric attacks often occur in the preataxic stage of posterior sclerosis and are attributed to all sorts of indigestion, but it is difficult to identify any actually determining cause. They sometimes occur but once in a given case. Ordinarily, they are repeated, and sometimes with regularity, every few weeks or months, or even daily. After several years they may diminish in frequency and definitely cease, or they may persist throughout the entire course of the disease. They may even cause a fatal termination.

Variations are not uncommonly encountered in which the gastric crisis may be unattended by much pain or the pain may be excessive, and vomiting slight or absent. They may closely simulate hepatic or nephritic colic, or be marked most by the generation of an extreme flatulency. Occasionally there is considerable blood in the vomitus, giving rise to the suspicion of gastric ulcer.

The intestine is often disturbed in tabes. In some cases there is persistent *careless diarrhoea*, marked by frequent slight liquid stools, not attended by colics or pain. In other instances *constipation* is beyond control and fecal accumulations, apparently even above the colon, cause distressing and persistent complaint. Intestinal or rectal *tension* occasionally drives the patient almost distracted. There is a constant desire to defecate, but efforts are abortive, or only result in a small passage. The repeated imperative character of these attacks, with their apparent carelessness, should serve to distinguish them. A rectal examination is usually negative. Rectal crises may simulate dysentery, particularly as considerable blood may appear in the stools. In many late cases the anal sphincter is incompetent to retain fluid bowel contents or injections.

The urinary apparatus presents some of the earliest indications of tabes. Among the first symptoms is a *difficulty in starting* the urinary discharge and in *completely evacuating* the bladder. This condition must be intelligently investigated, as it may escape the patient's attention or be deemed of no significance. As a rule, tabetics have to make forceful abdominal expulsive efforts to complete micturition, and in some cases this is aided by pressing the hands deeply into the lower belly-wall. Some patients can best or only urinate in the squatting position. Not infrequently after protracted waiting, the feeble stream sud-

denly ceases to flow, or after the act is thought to be completed a small amount of urine wets the clothing. Complete retention is rarely encountered, but daily catheterization may be required in some cases. Similarly, partial or complete incontinence is met with, but what is more common is an inability to control the escape of a few drops or more of urine if the slightest desire to urinate arises, or even the thought of it occurs. Many tabetics retain the urine well during the daytime and regularly wet the bed. Others, and this is common, feel no inclination to urinate, probably owing to some degree of vesical insensitiveness, and may only pass water once or twice in the twenty-four hours. Very frequent urination may be due to a weak *sphincter* or to *epithis*, which often arises from retention.

The *character of the urine* in tabes is frequently altered. *Glycosuria* is somewhat common among tabetics. The hereditary relation of tabes and diabetes has been already suggested. The medullary lesions of tabes may stand for something in this relation. In some cases there is a *quantitative diminution* of the urea, or phosphates, or chlorids. Sometimes the quantity of urine is notably lessened, sometimes greatly increased in a paroxysmal manner.

The mucous lining of the urethra and the bladder may be insensitive, but that does not prevent their being the seat of atrocious painful attacks that constitute *conical crises*, or, extending to the lumbar regions, suggest the term *septic crises*. During such attacks the patient is constantly tormented with the desire to urinate, but fails to express more than a drop or two at a time. Meantime the colicky, darting, intense pains about the neck of the bladder and down the urethra or thighs may give rise to intense suffering. Lightning pains in this region are not infrequent and vesical and rectal crises are often associated.

The generative functions frequently undergo modifications in tabes. In over one-half the cases there is a *loss of sexual appetite* and more or less *impotence*. Erections either completely default or are partial, and ejaculation does not take place. *Impotence* may develop very early, and sometimes is the first symptom to attract the patient's notice. In a majority of these cases there is an *early genital excitement* that has sometimes led to sexual excesses and has perhaps contributed to the idea of the *causal rôle* of such practices. The same thing is seen in general paresis. Other reflexes than the genital may be exaggerated in the initial period of tabes, as witness the increased knee-jerk that appears in very exceptional instances, and the spasmodic action of the bowels, rectum, and bladder.

With the loss of generative aptitudes in tabes we usually find a *diminution or complete extinction of the cremasteric reflex*. As there is a loss of appetite these patients make no complaints, contrasting sharply with the sexual neurosthenic, whose cremaster also is likely to be extremely active. In the same tabetic condition the *balloconvergent reflex of Oranoff*, or the *virile reflex*, as it is called by Hughes, is usually, if not always, absent. To secure this reflex the index-finger of the examiner is firmly placed over the bulbous portion of the urethra at the angle of the scrotum and perineum, and the mucous membrane of the urethral glands lightly pinched. The palpating finger will distinguish the contractions of the balloconvergent and ischioconvergent mus-



des. This reflex is said to be invariably present in healthy males, and even in other nervous diseases, whenever complete erection is possible.

*Testicular atrophy* has been already mentioned. It is present in about four-fifths of all tabetics, and is not infrequently attended by *atrophy of the testicle*.

In female tabetics there are analogous changes in the sexual sphere. In both sexes the genital organs are occasionally the site of painful crises that are commonly misunderstood, especially as they are prone to occur in the preataxic stage of the disease.

**The Respiratory Apparatus.**—*Hyperæsthesia* and *æsthesia* of the soft palate and diminution or increase of pharyngeal and laryngeal sensibility may be found separately or variously combined in cases of locomotor ataxia. Oppenheim has described *pharyngeal crises*, consisting of rapidly repeated, noisy, and very painful swallowing efforts that are involuntary. They last from a few minutes to a half-hour and are attended by some facial cyanosis and abundant perspiration. The writer has observed them in a case with progressive involvement of the cranial nuclei. A *spasmodic dry, barking cough* is not very uncommon.

*Laryngeal crises* are tolerably common in tabes, and vary greatly in degree and intensity in different cases. Sometimes there is a noisy, croupy inspiration, to which a cough may be added that strongly suggests whooping-cough. There is more or less dyspnea, pain, anxiety, and depression. In some instances the dyspnea seems to be absolute and the patient falls suddenly, cyanotic, unconscious, and convulsed. After a few moments the laryngeal spasm yields and full consciousness immediately returns. This form of laryngeal crisis has been called the *laryngeal stroke* by Charcot.

In patients subject to these crises they may be provoked by slight irritation of the laryngeal mucous membrane by mechanical or other means, and in some instances by pressure of a sensitive point on the side of the neck between the lower border of the larynx and the sternomastoid. Like other tabetic crises, they may increase in severity and frequency or grow less intense as time passes, and they leave the same tendency to recur. Though giving rise to alarming symptoms, they are, ordinarily, without danger in themselves. They appear to be due to unusual sensitiveness of the laryngeal surfaces.

*Laryngeal palsy* are encountered in tabes and may or may not be attended by laryngeal crises. The palsy may affect any of the laryngeal groups of muscles on one or both sides, but seems to exercise a preference for the dilators. Corresponding respiratory and vocal symptoms follow. The nerves, roots, and bulbar centers have been found variously degenerated, and the muscles themselves secondarily atrophied. *Bronchial attacks*, marked by spasmodic cough and respiratory difficulty, are rarely encountered.

**The Vascular Apparatus.**—Taking into view the fact that syphilis is one of the commonest causes of vascular deformities and disease, the frequency of arterial sclerosis and cardiac abnormalities in locomotor ataxia is not surprising. Aside from the cerebral vascular accidents arising from this source, angina pectoris, associated sometimes with gastric crises, is encountered. Valvular disease, affecting both the mitral apparatus, usually in the form of insufficiency, and the aortic valve,

mainly by stenosis, is found with considerable frequency. In 300 cases Limbach<sup>1</sup> found mitral insufficiency alone in 2 cases; with aortic stenosis once; 2 cases of aortic insufficiency were noted, 1 of aortic insufficiency and stenosis, and 1 aneurysm of the aorta—7 in all. Lesser<sup>2</sup> found aneurysm in 19 out of 96 cases, 20 per cent. A rapid pulse, from 100 to 120, is not uncommon. The blood may be normal or impoverished and frequently contains rhodin. It is almost invariably Wassermann positive.

The temperature in tabes is normal or only shows variations dependent upon intercurrent associated or secondary disorders. Pel<sup>3</sup> has reported a case with crises of high temperature and rapid pulse lasting about twenty-four hours. There were also coryza, lachrymation, photophobia, and lancinating pains in the face and eyes. Oppler<sup>4</sup> reports a similar case.

**Trophic Disorders.**—The disturbance of nutrition in tabes finds some manifestation in nearly every case, and there is no tissue or structure that may not be affected. The great majority of tabetics, and perhaps all in the later stages, show a deprivation of the *general nutrition* that can not be explained by their physical inactivity, by pains, or by a syphilitic osteitis. In the prestatic and static stages the general malnutrition is frequently well marked, but exceptionally the tabetic is plump, ruddy, and apparently vigorous.

**Ossous System.**—On the part of the skeleton tabetic dystrophy presents two striking clinical manifestations,—*spontaneous fractures* and *dystrophic arthropathies*.

**Spontaneous fractures** in tabes are more frequent than might be supposed, as they are not by any means always referred to their proper origin. It would appear that they are more common among women than in men. They may occur in the very early stages of posterior sclerosis, during the ataxic stage, or in the last phase of the disease. Their most frequent site is the femur, especially the shaft; the leg bones, and those of the forearm; but any long bone may present this accident, and even the vertebral bodies are sometimes thus affected. The fractures may be repeated or multiple in the same subject. Separations of epiphyses and of the bony insertions of muscles are also encountered.

Tabetic fractures are marked by the practical absence of pain in the affected part, and by the usually *insignificant* force that occasions them. They have followed merely the *crossing of the knees* or have taken place while the patients have been calmly walking on a smooth surface. Union takes place readily, but, owing to the lack of pain, which ordinarily reinforces the immobilization of the parts by splints, movements of the limb are not inhibited. *Slouching* and *contracture colles* are the natural results.

The bones in tabes and parietic dementia present a certain *fragility* which lays them liable to fracture during life. To the naked eye they often present a *porosity* and a shrinking of the compact substance, with an increased *sponginess* of the more open structures and sometimes an enlargement of the medullary canal. Microscopically, the Haversian canals are dilated and some decalcification is evident. The *osteoblasts*

<sup>1</sup> Deutsch. Zeit. f. Nervenheilk., 11, 1895.

<sup>2</sup> Berlin. Klin. Woch., Jan. 28, 1904.

<sup>3</sup> Berlin. med. Woch., Jan. 26, 1900.

<sup>4</sup> Berlin. Klin. Woch., 11, 1902.



are shrunken and sometimes show fatty degeneration, while the medullary substance is increased to an amount corresponding to the diminished osseal portion. This results in an inversion of the ordinary ratio between the organic and inorganic elements. Normal bones are about two-thirds inorganic substance; tabetic bones are about *two-thirds organic matter*. In other words, these bones present a *calcifying osteitis*. By some this is attributed to changes in the nutrient artery and nerve, which have been found sclerotic and neuritic. For others the lesion is a manifestation of the trophic disturbance arising from modification of trophic cells in the spinal ganglia and root.



Fig. 192.—Spontaneous fracture and osteomyelitis (tabes dorsalis).

**Tabetic Arthropathy.**—One of the early features of tabes is an *obscure stage of joint-lesion*. This may be observed even in the pre-ataxic stage, but is usually developed after incontinence has appeared. Parnau<sup>1</sup> was among the first to call attention to the fact that the joints in tabetics could be forced into extreme flexion or extension without producing much or any pain, and attributed the fact to analgesia. Fränkel and Paure,<sup>2</sup> in a fuller study of the matter, show the extreme

<sup>1</sup>"*Bost. M. and S. Jour.*," Aug. 29, 1886.

<sup>2</sup>"*Nouv. Ann. de la Salpêtr.*," July, 1896.

range of motion that is customarily found in the joints in this disease. They found that in locomotor ataxics the foot, wrist, elbows, and fingers are similarly affected. Their patients could execute at the first attempt the "split" attitude that acrobats only attain by years of practice. (See Figs. 188, 189, 190.) They attribute this abnormal flexibility to muscular and joint analgesia in part, but principally to a loss of the muscular tone. This condition of the joints places them at a certain disadvantage, so that the joint-surfaces are not properly coapted and ligamentous stretching is often induced. Taken with the incoördination of movement, there is little doubt that the joints are subjected to unusual traumatic twistings and shocks. In addition, there is present the trophic disturbance marked by fragility of bones, and out of it all arises the *tubercle* or *Charcot's joint*. Its frequency in syringomyelia adds force to the belief that the spinal lesion is the principal cause, the traumatism the excitant.



FIG. 191.—Tubercle ankylosis of the left knee.

A *tubercle ankylosis* is marked at first by (1) rapid or even sudden onset, (2) entire or nearly entire absence of pain and tenderness, and (3) excessive swelling of the adjoining parts. The patient, while walking or using a member, may notice a sharp crackling in a joint and find that the limb feels heavy and more unmanageable than usual. Shortly—that is, in the course of a few days—not only the joint, but the entire segment of the limb is greatly swollen. This swelling may be the first thing to attract attention. The parts are found tense but cool, and devoid of



redness and tenderness. The swelling is extreme, but not boggy, and does not pit on pressure. Movements of the joint and its employment in no way inconvenience the patient. In *favorable cases*, after a little time, the swelling subsides and all trouble disappears except a little thickening about the joint and some creaking in the articulation. There is, however, a great tendency to the recurrence of aggravated and more lasting attacks. In *severe cases* the swelling does not disappear so promptly, but becomes circumscribed about the joint in a more or less *globular form*, and the joint-surfaces as well as the ligamentous structures undergo *disintegration*. Finally, the limb-segments may be united only by soft tissues that permit painless motion and circumduction in every direction. Added to the joint-changes, we may have spontaneous fractures, epiphyseal separations, suppuration, and even the protrusion of the bones through the skin. Old tabetic joints present merely a bag of bone-fragments where articulations were formerly located.

In 132 cases of tabetic joint disease Krodol found arthropathies occurring 21 times in the prodromic or preataxic stage, 38 times between the first and fifth year of the disease, 32 times from the fifth to the tenth year, and 41 times after the tenth year. They occur in 3 or 4 per cent. of all cases, and more frequently in women than in men.

The localization of joint-disease in tabes is mainly in the large joints, but no articulation is exempt. Flatau collected 139 cases, in 41 of which there were bilateral arthropathies. The order of frequency was as follows: knee, 60; foot, 39; hip, 38; shoulder, 27; elbow, hand, fingers, and maxilla, 4 to 6 times each. The plantar arch usually yields, and *flat-foot* is the rule in tabes.

Upon section of these joints the *capsulae* are found dilated, often ruptured, and in old cases completely destroyed. The *ligaments*, especially those within the joints, as at the knee and hip, are diseased or have disappeared. The *synovial membrane* is thick, rough, and often adherent to the surrounding parts; later it may be absent. It may contain bony particles and osseous nodules. The *joint-fluid* is thin and clear or yellowish and exceptionally purulent or bloody. At first it is abundant, and infiltrates the parts about the joint, into which it escapes through the ruptured capsule, and accounts in some cases for the great swelling in the limb. It may contain floating bodies in large number, bony particles, and detritus.

The ends of the *bones* and joint-surfaces may be either (1) eroded, as is most usual, and greatly reduced in all their dimensions, even to the complete destruction of several inches of their length, or (2) may present the hypertrophic exaggeration of an arthritis deformans. These two types may be combined in the same joint. The rule is that the



Fig. 104.—Tabetic arthropathy of right knee, with atrophy of the entire extremity (Krodol) and J. D. Chace.

hypertrophic form occurs in the knee, the atrophic variety at the hip and shoulder. The disintegration of the joint may be increased by intracapsular fractures or by fragmentation of the eroded shafts and separated epiphyses.

Juergens has found that in most tabetics nearly all the joints show capsular enlargement, elongation of ligaments, vascular dilatation, and some synovial roughening. The preponderance of such joint-disease in



Fig. 100.—(1) from top, 2 and 3, show deformity due to atrophied disintegration of the tarsus; 2 has a perforating ulcer under head of 5th metatarsal.

the lower extremities depends, perhaps, upon their more exacting and vigorous use, and consequent liability to strains and traumatism and upon the major lesions of the lumbar cord.

**Trophic Cutaneous Disorders.**—In tabes there are a number of trophic dermaloses that are of rare occurrence and insignificant importance. *Herpes zoster* is of more frequent appearance, and finds its favorite location on the trunk, rarely in the distribution of the trifacial. The epidermis of the extremities, especially the upper ones, is sometimes hypertrophic. *Hyperidrosis*, *hidrosis*, and the loss of nails or teeth have been occasionally noted.

*Perforating ulcer* is not an uncommon accident in tabes, and, though painless, is of considerable importance. It is usually situated in the foot, but may occur in the hand, and some authors have conceived that maxillary and even cardiac and visceral ulcerations were in some instances of the same character. It usually begins as a callus or corn on the sole of the foot, under the ball or under the base of the fifth metatarsal, or at the heel. Ulceration follows, and, if neglected, may denude bone and lead to exfoliation. The ulcer is indolent, persistent, and refractory to any treatment if pressure be not removed. Not uncommonly the toe-joints or those of the foot present atrophic conditions at the same time. *Blebs* only appear in terminal stages, and present nothing of a special nature. After an attack of lightning pain the part in which they are principally located may sometimes present a more or less distinct *eczymosis* or *edema*.

**Muscular Atrophies.**—In addition to the invariably diminished muscular tone that has already been mentioned, and the rare occurrence of *feather of a tendon*, some tabetic cases present notable *myotrophies*. This should be sharply distinguished from the emaciation, flaccidity, and incoordinate feebleness that are very common in advanced tabes. As a rule, the muscular masses and contours are well preserved until the



staxia is well developed, and often until the patient has for long been unable to walk, but in rare cases, perhaps in one per cent., localized muscular atrophy appears. Its common seat is in the lower extremities, especially invading the foot- and leg-muscles, and is usually bilateral. The upper extremities may be invaded, particularly the small muscles of the hands, or even the forearm, arm, and shoulder. The cranial nerves are sometimes similarly affected. The motor portion of the trifacial and the hypoglossus are the ones usually selected.

The onset of such atrophies is usually insidious. They present variously modified electrical reactions, the full reaction of degeneration being rare. Once established, the muscular atrophy of tubes remains fixed, and does not invade group after group of muscles, as do various progressive atrophies, but in very rare cases a rapid and extensive general muscular atrophy is encountered that reduces the patient to skeletal proportions and total disability. The resulting deformity in the foot is due to pure, flaccid atony without contracture. The foot drops by its own weight and the pressure of bed-covering into an equinovarus. In the hands some clawing may be induced and the thenar eminence is likely to disappear. Hemiatrophy of the tongue follows hypoglossal involvement.

In a general way, according to Marie, tabetic muscular atrophies may be divided into two groups: (1) Those appearing at an advanced period of the disease, presenting a symmetrical distribution, rarely marked by fibrillary twitchings; (2) those occurring often in the earlier stages of the disease, usually unilateral in distribution, and marked by fibrillary contractures and sometimes by the reaction of degeneration. The first group embraces those atrophies confined to the distal portions of the lower or upper extremities, and recalls the conditions found in multiple neuritis. The second group contains lingual hemiatrophy, localized atrophies of the shoulder, of the back, of the hand, and one-sided involvement of cranial nerves. They are analogous to lesions of the nuclear gray matter. Both the central and peripheral lesions are found, and in the associations above indicated. The wasted muscles present the usual histological change, due to degeneration in the lower motor neuron.

**Cerebral Disturbances.**—In addition to the vascular cerebral accidents, with resultant palsies and the involvement of cranial nuclei, tabetics are subject to other cerebral disorders. These embrace the many possibilities of cerebral syphilis, and particularly parietic dementia. Apoplectiform and epileptiform attacks, or any unusual forgetfulness, exaltation, expansiveness, or stupor should at once arouse suspicion of this fatal cerebral disorder. It should be studied in this connection, and is set forth in the second part of this work. On the whole, notable psychical disturbance is a rarity, but some degree of apathy, of indifference, is usually to be observed. This pertains particularly to themselves, their disease, their almost hopeless prospects. It is not much modified, even by the most atrocious suffering, and persists even in the stage of complete helplessness. Tabetics, however, often manifest large mental activities and retain their business capacity to the end.

**Tabulation of Tabetic Symptoms.**—The following table of tabetic

symptomatology is that of Limbich,<sup>1</sup> based upon 400 cases selected from the private practice of Professor Erb.

In the first table the usual early symptoms are arranged in their order of frequency. There is an overlapping, as frequently two or more are alleged to have come on at or about the same time.

#### EARLY SYMPTOMS OF TABES.

	First Symptom	Second Symptom	Total
Lancinating pains . . . . .	283 times	52 times	335 times
Cytic weakness . . . . .	91 "	119 "	209 "
Feeling of weakness in legs . . . .	78 "	112 "	190 "
Paresthesia in legs . . . . .	74 "	10 "	84 "
Girdle sensation . . . . .	34 "	44 "	78 "

The relative frequency of various objective and subjective symptoms as observed in these 400 more or less complete histories is shown in the following table:

#### RELATIVE FREQUENCY OF TABESIC SYMPTOMS.

	PERCENT.
1. (a) Failure of knee-jerk and Achilles jerk . . . . .	92.0
(b) Alteration in these reflexes . . . . .	4.25
2. Staring with eyes closed . . . . .	88.75
3. Lightning pains . . . . .	88.25
4. Disturbances of the bladder . . . . .	80.5
5. Ataxia of the lower extremities . . . . .	74.75
6. Changes in the pupillary reactions . . . . .	70.25
7. Paresthesia of lower extremities . . . . .	61.5
8. Feeling of weakness in the legs . . . . .	62.25
9. Diminution or disappearance of sexual desire . . . . .	58.25
10. Alteration in size of pupils . . . . .	48.25
11. Delayed conduction of pain . . . . .	36.5
12. Slight analgesia of lower extremities . . . . .	33.75
13. Girdle sensation . . . . .	31.0
14. Transitory double vision . . . . .	26.5
15. Distortion of sense of touch on lower extremities . . . . .	23.25
16. Paresthesia in clear distribution . . . . .	16.5
17. Ocular palsies and ptosis . . . . .	16.0
18. Optic atrophy . . . . .	6.75
19. Persistence of painful impression in the legs . . . . .	5.0
20. Various crises . . . . .	3.25
21. Arthralgias . . . . .	1.75

**Course and Varieties.**—Ordinarily speaking, the onset of tabes is extremely insidious and its course very slowly progressive. For purposes mainly of description it may be divided into the *prostatic*, the *atonic*, and the *paralytic* stages. These indefinitely blend, and, as has been repeatedly indicated, many symptoms, commonly of the later periods, may appear precociously in the early phases of the malady. From the tabulation of symptoms, as well as from their individual description, it will have been noted that pains are among the earliest indications of tabes, and these may persist for years, even for a dozen years, before the prominence of other symptoms determines their character. Usually only when visceral crises, visceral weakness, ocular palsies, insecurity upon the legs, or inability to walk in the dark or down a stair or to stand securely while washing the face have seriously attracted the patient's attention is a properly directed investigation instituted.

<sup>1</sup>Deutsche Zeit. f. Nervenheilk., 1905.



Then his "rheumatic pains," his "gouty pains," his "neuralgic attacks," his "biliary attacks," take their proper place. At that time a search of the cutaneous sensibility usually reveals its impairment in the feet and legs, the knee-jerks are absent, the pupils sluggish or inactive to light, and the ataxia can be demonstrated by the usual tests. In certain rare benign cases the disease never progresses beyond this point.

In the *second* period the ataxia increases and is apparent at a glance, but may be practically confined to the lower extremities for from two to six years or more. Then it may invade the upper extremities progressively. A host of sensory, motor, trophic, and visceral symptoms are present, varying in every case but usually consistent and uniform in the given instances. Even at this point the disease may halt in its progressive course. Usually the lack of motor control becomes greater and greater, walking more and more laborious, the ataxia intensified, and finally the patient is brought to the bed or chair in the *third* period of the disease. Now accentuation of intestinal and especially of vesical disturbance and the depreciation of the general physical state, taken with the helplessness, make the picture pitiful indeed. All its colors may be deepened by the atrocious pains that sometimes pursue the unhappy victim to the last. Cystitis looms as a constant menace to life, and any intercurrent affection is likely to be promptly fatal. From ten to twenty or thirty years may be consumed in the history of tabes or it may unroll its panorama of symptoms within two or three.

Tabes presents numerous variations from the wide symptom group that may be considered its common type. The *cervical form* presents pain and ataxia first in the upper extremities, which may also show trophic changes. Little static ataxia or incoordination of locomotion may be presented. The knee-jerk may even be retained, but that is rare. In the *bulbar form* we encounter early symptoms on the part of the cranial nerves, pharyngeal and laryngeal crises, optic atrophy, and ocular palsies. The tendency of ataxia to disappear upon the appearance of optic atrophy or for the disease to then become stationary furnishes a definite group of cases.

From another view-point, cases may be considered *benign* and *grave*. As has been indicated, the tabetic process may stop at almost any point, or after a lapse of years may again slowly advance. Some cases that are marked by intensely painful manifestations seem to be of slow evolution. This may be a way of saying that cases presenting a protracted first and second stage, to which the lightning pains and intense crises are usually confined, less rapidly disable the patient. On the other hand, cases of tabes are grave by the rapidity of their development and the intensity and generalization of their symptoms, due to the wide-spread underlying sclerotic process. Acute cases may confine the patient to bed in a few months. Leyden describes cases of extreme rapidity. Many times after a long, nearly stationary period there is a sudden increase of ataxia not attributable to any physical cause, or again, undoubtedly induced by strain, trauma, or illness. Any patient confined to bed for a few weeks is likely to be made much more ataxic by such restraint. Active syphilitic processes in brain and cord may take place, and the patient be overthrown at once. A marasmod state or the appearance of parietic dementia constitute conditions of extreme gravity.

Juvenile tabes occurring in youth between six and twenty-six years of age, in the subjects of parental syphilis generally, very much less frequently secondary to early syphilitic infection, is a tolerable rare form of this disease. Such instances commonly also show a neuropathic makeup or inheritance. Hirtz and Lemaire<sup>1</sup> show from a study of 47 cases as recorded in the literature that some clinical peculiarities are commonly encountered. The malady usually begins with urinary troubles, less frequently with lightning pains, least frequently with amblyopia. Gastric and intestinal crises early are frequently noted. The incoordination is relatively slight and tardy in appearing, marked ataxia is unusual. Pupillary signs and abolition of tendon reflexes occur with the same frequency as in adult cases. Laxaron,<sup>2</sup> following von Halban, calls attention to trigeminal headaches as of frequent occurrence in the early periods. The clinical type is not infrequently obscured by the presence of active or gross syphilitic lesions of brain and cord, by mental and physical defects due to the same cause, or by bodily infirmities to which such individuals are liable. The stigmata of hereditary syphilis can usually be easily noted.

**Diagnosis.**—The diagnosis of tabes in the full blown ataxic stage rarely presents any considerable difficulty. Confusion usually arises by mistaking other diseases for tabes and in misinterpreting the early manifestations of tabes for those of slighter ailments. Gastric, laryngeal, intestinal, vesical, urethral, and all visceral crises, if present in the preataxic period, are almost invariably referred to the wrong source. Their repetition without clearly competent exciting cause or local lesions should always arouse suspicion of posterior sclerosis, which, if present, will not fail to present other symptoms and signs. The same is true of repeated attacks of severe pains of a lightning or lightning-like character. The occurrence of these in a patient where syphilis is even suspected to have been present should direct attention to the spinal cord.

The frequency with which tabes is confused with other painful diseases is graphically shown by Nissim,<sup>3</sup> who found that 95 useless surgical operations had been performed in 1000 cases of locomotor ataxia. Gastric ulcer, gall-stones, appendicitis, salpingitis, renal calculi, and several other intra-abdominal disorders had been suspected. Exploratory laparotomy had been done nine times. In every case of repeated abdominal pains it would be a quite safe procedure for the surgeon to exclude tabes.

If the knee-jerks are gone or very unequal, or even greatly reduced, it should add to the suspicion of tabes. In early cases the condition of the heel-jerks is often of significance, as they tend to disappear even before the knee-jerks are affected. If, now, the Robertson pupil is detected, or even sluggishness of the pupil to light is clearly made out, the diagnosis may be considered established. The detection of several or many of the usual subjective and objective features of the disease will confirm it. Among these, too much importance can not be given to vesical disturbances and variations in the sexual sphere. The determination of lymphocytosis of the spinal fluid, with an increased globulin, and the Wassermann positive in blood or spinal fluid, all have great significance in doubtful cases.

<sup>1</sup> *Rev. Neurologique*, March, 1905. <sup>2</sup> *Neurolog. Centralbl.*, Nov. 10, 1905.

<sup>3</sup> *Jour. Amer. Med. Assoc.*, February 12, 1916.



The condition most usually mistaken for tabes is multiple neuritis. The differential indications are tabulated on page 332. Unfortunately, a group of maladies of a similar, if not identical, character with multiple neuritis has been denominated *pseudotabes*. We thus encounter cases described as toxic pseudotabes or *neurorabes*, due to alcohol, arsenic, or other poison, diabetic pseudotabes, neurasthenic pseudotabes, and syphilitic pseudotabes. They often present the symptoms of multiple neuritis, with unusually severe root pains, ocular disturbances, or other symptoms that suggest tabes. The absence of well-marked crises, of the Robertson pupil, of sphincteric weakness, of pure incoördination without paresis, and the history of the onset of the disease, the uniformity and invariability of sensory symptoms, the history or presence of the toxic cause, and the usual early presence of some muscular wasting and the reaction of degeneration should distinguish pseudotabes from posterior sclerosis.

*Paraplegia* are marked usually by definite segmental areas of dysesthesia, the reflexes are exaggerated, and clonus common, unless the cord is completely divided, when all motion below the lesion is abolished. This is not the case up to the last moment in tabes.

*Cerebellar disease* may present some analogy at first sight, but usually we have choked optic discs, increased or merely reduced reflexes, intact sensibility, retracted head, and occipital pain.

*Inular sclerosis* presents some symptoms found in tabes, but is distinguished by the intention tremor, nystagmus, scanning speech, usually preserved and often increased knee-jerks. The sensory disturbance and painful manifestations are insignificant. The spinal fluid is negative in the non-syphilitic.

*Syringomyelia* usually affects the upper extremities first, and may be mistaken for cervical tabes. The dissociation of cutaneous sensibility is its chief characteristic, but even this has been found in tabes. Scoliosis, mutilations of the fingers, local atrophies occurring early in the disease, without incoördination and usually with increased knee-jerks, taken with a full history of the case and the careful delimitation of the sensory disturbances, should make the diagnosis exact.

Finally, a careful examination of the *spinal fluid* withdrawn by Quincke's puncture may furnish conclusive evidence.

**Prognosis.**—The diagnosis of tabes largely conveys the prognosis. When the degenerative changes that constitute the so-called sclerosis have taken place, restitution of *integrum* is, as far as now known, out of the range of possibilities. While, as a general rule, the disease is a steadily progressive one, there are many exceptions. In the enumeration of varieties of tabes attention was called to the benign cases and the possibility of the degenerative changes coming to a standstill at any period of development. This renders it the more difficult to estimate the value of medication. It has also been indicated that when optic atrophy appears the locomotor difficulties usually do not increase. The rate of advancement of the disease from its inception is some index of the rapidity of its future progress. Bulbar symptoms and the indications of developing parietic dementia at once render the outlook most gloomy. The duration of life is not, however, abridged as much as might be supposed. Marie and Magnat,<sup>1</sup> on a basis of 66 patients who

<sup>1</sup>"Sem. Méd." 1903.

had died at the Bichat, found that 55 had passed the fiftieth year and 34 had lived beyond sixty. The cause of death is usually some intercurrent affection not necessarily associated with tabes, though vesical inflammation and secondary infection of the kidneys are always to be apprehended.

**Treatment.**—In the management of tabes dorsalis it is well to keep in view exactly what may be accomplished. The retardation of the disease, or, better still, its complete arrest, constitutes a medical victory. Given the natural tendency of the disease to halt temporarily in occasional cases or to come to a permanent standstill, we must be chary of attributing too much importance to any line of medication. The intelligent purpose of treatment is: (1) To arrest any active syphilitic process that may be present; (2) to improve the general health; (3) to increase the nutritive condition of the cord; (4) to maintain as complete muscular control as possible, and (5) to meet the host of incidental disturbances as they arise.

While the rôle of syphilis in tabes is chiefly played in the past tense, it not rarely happens that active and manageable syphilitic lesions attend at least its early stages. Meningitis, myelitis, neuritis, and cerebral conditions or affections of the osseous system may declare themselves, and yield to antisypilitics. The actual presence of the spirochete in the lesions of tabes gives substantial reason for antisypilitic medication. Numerous French and German writers strongly recommended intensive mercurial treatment with alleged good results in arresting or improving the disease. Intramuscular injections of soluble or insoluble preparations of mercury are used. Brockhart<sup>1</sup> thus treated 58 early cases, securing improvement in 33 and recession of the disease in 12. Lemoine, Devy, and Lerrôle have had similar results. The author feels confident that he has arrested optic atrophy in several advanced cases by this method.

Salvarsan, with or followed by mercury, may be used as long as Wassermann's blood test gives a positive reaction. The intradural administration of neosalvarsan, either by the autoserum method of Swift and Ellis, of the Rockefeller Institute, or some of its modifications, gives little hope of more efficiently reaching the spirochete than can be done by intravenous or intramuscular methods. In rare cases any form of intensive antisypilitic treatment will secure negative serological reactions, but in the great majority no amount of arsenical or mercurial treatment by any or all methods produces this desirable result. As long as the blood remains Wassermann positive courses of treatment should be persistently carried out. Thereafter, guided by the condition of the spinal fluid, periods of intensive treatment should be employed at intervals of three to twelve months, depending upon the clinical manifestations and the general results. The iodids are not recommended or used by the author at any stage of tabes.

The general health, with all the conditions of hygiene, good air, and a proper diet that pertains to it, are worthy of painstaking attention. Not only does a good general state tend to retard the activity of tabes, but it protects the patient from the great dangers of intercurrent affec-

<sup>1</sup> *Monatsh. für praktische Dermatol.*, 1902.



tions, especially of the acute variety. The patient must guard against physical strains and exhausting effort of every sort.

The local nutrition of the cord and spinal apparatus may possibly be improved by increasing its blood-supply. This may be mechanically effected by spinal stretching. It is not desirable to carry this out, as first was done, by hanging in the Sayre apparatus—a proceeding that is attended by a number of dangers. It can be effectively accomplished by having, after Benedict's suggestion, the patient fully flexed upon himself. Teuette and Chipault<sup>1</sup> have proved that the lower portion of the cord may actually be stretched by the forward bending of the trunk. Care must be exercised not to overstretch the patient at first, as the thigh and back muscles may easily be severely strained. Gradually, in the course of a week or two, through daily stances, the full body flexion, and consequent extension of the cord, can usually be attained without inconvenience. The flexed posture is to be maintained for two or three minutes only, and may be utilized night and morning. It often favorably modifies the lightning pains, and sometimes increases the sexual aptitude. The stretching is accomplished by seating the patient on a low table or on the floor, with the lower limbs extended. The head is then forcibly depressed toward the knees, which are not allowed to bend. The position is to be maintained not to exceed two minutes. Patients, after a little instruction, can carry out this plan themselves.

Local measures to the back, such as deep massage and very vigorous tapping, are of similar benefit to the deeper circulation. For this purpose a broad, stout piece of leather on a short handle may be used to vigorously flagellate the back. Its similar application to the soles of the feet and on other paræsthetic or analgesic regions is useful. Counter-irritants along the spine have a certain value, perhaps only an insignificant one. The best application is by the thermocautery of Paquelin, repeated every seven or ten days. An intense white heat should be used, and small dots quickly and lightly made at intervals of an inch or two over the portion of the cord principally affected. They may with propriety be extended over the course of the nerves where the lightning pains discharge. Six, eight, or ten such cauterizations may be followed by two or three months' rest, and then repeated. Cold spinal douches, hot needle douches, or steam douches to the back are of comparable utility.

It is only of late that the value of exercises and practice to reestablish coordination has been recognized. On the other hand, inactivity and disuse promptly accentuate the loss of muscular control. The purpose is not to accomplish feats of strength, and all strains and decided fatigue are to be resolutely avoided. Patients must be encouraged to faithfully and intelligently practise such movements, motions, and steps as are particularly uncertain. In this way they may sometimes be brought to stand and to walk with closed eyes after static ataxia has been well marked. As many ataxies experience no feeling of fatigue, watchfulness on the part of the physician is imperative. It is a point of practical importance to see that the patients have proper shoes that support the yielding and frequently badly broken tarsal arch. Frankel<sup>2</sup>

<sup>1</sup> *Nouv. Icon. de la Salpêtrière*, June, 1907.

<sup>2</sup> *The Treatment of Tabetic Ataxia*, Philadelphia, 1902.

gives the following exercises, which are of two classes—those performed in and those performed out of bed, depending on the patient's helplessness:

In bed, the patient is called upon to flex, extend, abduct, and adduct each leg separately and then both simultaneously. The knees and hips are likewise exercised. The patient is asked to place the ball of one foot on the big toe of the other foot, to place the heel upon the knee of the other leg, and then slowly travel along the ridge of the tibia toward the ankle. Exercises are made alternately, first with one leg, then with the other, with open and with closed eyes. These exercises are attempted over and over again, with frequent rests. The patient is encouraged to persevere until he succeeds.

The exercises are repeated twice a day, a half hour in the morning and again a half hour in the afternoon if the patient's condition justifies it.

1. Patient is placed with his back to a chair, heels together, then seats himself slowly in the chair, and is then made to rise in the same careful manner. No cane is used. If patient can not stand, an attendant is placed on either side to support him if necessary.

2. One leg is placed at an ordinary walking step in front of the other, and then placed with great exactness back into its original position. Same exercise is then performed with other leg. The patient, if necessary, supports himself by a cane or otherwise.

3. Walks three paces slowly and with precision.

4. Rest in standing position, one foot before the other; with hands placed skilfully he flexes his knees and slowly raises himself again.

5. Patient, as in exercise number 2, advances one foot, then returns it to its original position, and then places it one step behind the other. This exercise is usually a very difficult one, requiring, as it does, a great deal of balancing power.

6. Walk twenty steps as in exercise number 3.

7. Number 2 performed without a cane.

8. Stand without a cane, with feet placed together and hands on hips.

9. Stand without a cane, feet separated; various movements with the arms, grasping objects, forcing back outstretched hand of physician, etc.

10. Maintain same position as in number 9, flexing trunk forward, backward, to the right, and to the left.

11. Exercise number 9 with the feet together.

12. Exercise number 10 with feet together.

13. Walk along a painted line on the floor, patient supported by a cane.

14. Same without a cane.

Exercises for the fingers and arms are also employed based on the above-mentioned principles.

These various exercises are to be progressively attempted and persevered in as coordinate strength improves. They may then be gone over again with closed eyes aided by a cane or assistant, then without aid. Fatigue, however, must be avoided. In advanced cases that have to rely on crutches, a tall "walking-frame" or roller-catch, such as is used for children, may be employed. This gives support under the arms and enables the patient to exercise the legs. Precise, delicate motion with the fingers may also be developed in the same way. The suppy-



ment of an intelligent masseur has given good results in some instances, as he can, by the use of graduated resistive movements, teach the patient precision in the use of his legs. The so-called home bicycle trainer has also given some assistance. On this the patient can pedal for a few minutes at a time, and the mechanism carries his feet and legs in definite curves.

In the pre-ataxic stage the patient should walk, stand, and balance on each foot, with closed eyes. Daily exercises of this simple character seem notably to retard the appearance of locomotor incoordination. Women are much less troubled than men in the control of the legs and feet, as a rule, and this may be due to wearing skirts, which prevents the use of the eyes in guiding the steps. The lack of ataxia in the early blind cases is also significant in this connection.

Regarding *internal sedation* directed to the sclerosis, mercury, arsenic, silver, chlorid of gold, salts of zinc, strychnia, aconitin, atropin, and a multitude of others may be mentioned, but aside from some general tonic properties it is difficult to attribute any value to them. Ergot, first employed against a hypothetical chronic inflammation in which the sclerosis was supposed to consist, has proved itself of some value in controlling vesical disturbances, and against this feature of tabes may be employed, with precautions to avoid ergotism. Charcot's plan was to use it the first three days of every week, or it may be used on alternate weeks for one or two months, then a long interval and a repetition. It should be used in good-sized doses once or twice a day.

Against the *coital weakness*, especially the sphincteric weakness, the method of Bismitt to increase the strength of the pelvic floor is of service in tabes. The movements principally useful consist of having the patient, while lying on the back, separate and adduct the knees against resistance from two to twenty times twice daily, at the same time vigorously drawing in the pelvic floor and body outlets. Massage of the perineal muscles may be helpful.

For the *peas of tabes* and the *visceral crises* morphia is sometimes required, but the physician should administer it, to prevent the formation of a habit, and then only as a last resort. Iod, hot applications, sinapisms, and the coal-tar sedatives should be first thoroughly tried. Of the synthetic preparations, phenacetin and aspirin seem the most efficient. Blisters and the cautery to the painful region and over the corresponding portions of the cord are sometimes promptly helpful, but must be used with circumspection, as healing is often faulty. Resection of the posterior roots of the seventh to tenth dorsal nerves in otherwise unmanageable cases of continuous gastric crises have been favorably reported by Foerster and Küttner, Bruns and Sauerbruch, and also in this country. Similar operations may be considered as a last resort in critical attacks elsewhere situated.

Tabetic joints are best let alone. Nothing is to be gained by cutting operations, and very little by fixation apparatus except such as enable the patient to walk. Perforating ulcer is sometimes cured by stretching the nerve to the part.

*Cystitis* must be guarded against. If it develops, it must be carefully controlled, and self-catheterization may have to be taught the patient. Urotrin in ten-grain doses, twice or thrice daily, may be employed

for indefinite periods to keep the urine aseptic and prevent cystitis, but sometimes irritates the kidneys.

The management of the individual will often be found as difficult as that of the disease. He must not expect too much, but the physician must remember that he is human, and do what he can to encourage and cheer him in the face of his distressing affliction, and to insure his faithful attention to the numerous small and exacting details of treatment.

The appearance of any active syphilitic process demands prompt recurrence to specific treatment.

#### COMBINED SCLEROSIS OF THE SPINAL CORD.

Occurring, perhaps, as frequently as tubercles, many cases of organic cord-disease present symptoms referable to the simultaneous involvement of the lateral and the posterior columns, which are found sclerosed in widely varying proportion. This condition is termed *chronic paraplegia* by Gowers, *progressive spastic ataxia* by Dana, and is known variously as *posterolateral sclerosis*, *combined posterior and lateral sclerosis*, and *combined tabes*, or sometimes as *syssostic tabes*. Some cases show a tolerably definite limitation of the sclerosis, suggesting a systematic degeneration, but usually it is not strictly confined to the physiological tracts of the cord. For the most part the lesions are within the posterior half of the cord's cross-section, and are commonly embraced in the posterior arterial field (see p. 345 and Fig. 120). Occasionally the lesion also encroaches upon the peripheral portion of the cord in front, which belongs to the anterior arterial field. The symptoms are dominated by ataxia and spasticity, and the tendency is progressively to a paraplegic helplessness.

**Etiology.**—The causes of combined sclerosis are numerous. It must be at once recognized that this extensive symptom group is frequently a secondary (end-process) (1) In a certain small number of cases, primarily tabetic, a diffuse myelitis also involves the lateral tracts; (2) it

is found more frequently in general paresis than are lesions entirely confined to the posterior columns; (3) a diffuse myelitis gives rise to ascending and descending degenerations that furnish a posterolateral sclerosis; (4) leptomeningitis may entail a marginal myelitis that involves the periphery of the cord and principally affects its posterior half; (5) vascular lesions, affecting principally the posterior field, induce a sclerosis in the posterior and lateral tracts; (6) pellagra sometimes causes a combined posterolateral sclerosis; (7) toxic

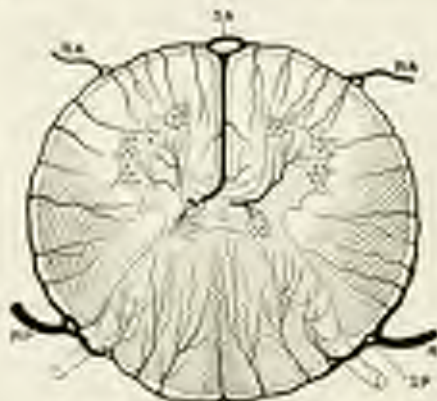


FIG. 120.—Combined posterolateral sclerosis. Shaded showing small area of involvement limited to the posterior arterial field of the cord (Elliott).

conditions, as in the pernicious anemias, may cause it; (8) the posterolateral sclerosis may be evidence of an endocrine deficiency as in Friedreich's ataxia.



There is frequently a history of antecedent syphilis or one of exposure to cold, traumatism, contusion, or muscular strains. Sometimes acute infections lead to it. It is much more common in males than in females, and usually appears between twenty-five and forty years of age.

**Morbid Anatomy.**—The sclerosis implicates both the lateral and posterior columns, but sometimes one, sometimes the other with greater intensity. The *columns of Goll*, usually commencing above the lumbar enlargement, are degenerated to the medulla. The *columns of Burdach* are also affected mainly above the lumbar enlargement, but less intensely, and the root-zone usually escapes. The *crossed pyramidal tract* is, as a rule, partially affected, its inner portion being customarily spared. Its lesion is slight in the cervical region, and increases in the lower dorsal and lumbar portions of the cord. The *direct pyramidal tract* is only affected when the lesions are intense in the upper cervical region, and even then but slightly. The *direct cerebellar tract* is always intensely affected. The ascending tract of *Goerz* sometimes escapes, sometimes is partially affected. The *gray substance* is usually intact. Rarely the anterior cornual cells or those of the posterior horn or of Clarke's column may be slightly modified.

The sclerotic areas differ from the degenerated portions of a tabetic cord in presenting hypertrophied axes-cylinders, many spider-cells, and notable changes in the blood-vessels. The alteration in the nervous tissue is also proportionate to the vascular lesions and most intense in their neighborhood. Occasionally a chronic meningitis is present. Cranial nuclear involvement has been found in some cases.

To sum up, the lesion is of a myelitic character rather than that of a pure parenchymatous Wallerian degeneration. It affects the posterior columns from the lower dorsal area upward and the lateral tracts from the cervical region downward in increasing intensity, precisely as do secondary degenerations. The lesion is indeterminate in localization and bears a close relation to the arterial supply of the part.

**Symptoms.**—The symptoms of the combined sclerosis are those of spastic paraplegia and locomotor ataxia variously combined and associated. One case shows a preponderance of ataxia, another of spasticity, depending upon the distribution and intensity of the lesion in the posterior or lateral columns and upon its vertical extent. Lesions of these tracts give us, on the part of the posterior columns, incoordination, an ataxic gait, Romberg's sign, diminished or lost reflexes, lancinating pains, anesthetics and paresthetics, ocular symptoms, vesical and genital derangements, etc. Related to the lesion of the lateral tracts we have exaggeration of tendon reflexes, foot-clonus, Babinski's toe-sign, motor encephalogram, cramps, spasms, and the spastic gait. By their association in posterolateral sclerosis we usually find ataxia and spastic weakness, with increased tendon reflexes, well marked in the lower limbs, less pronounced in the upper extremities, and cranial-nerve symptoms are seldom entirely absent.

The onset of the disease, when not due to a diffuse myelitis of more or less acute character, is insidious. One of the earliest complaints is that of becoming readily tired in walking, the legs feeling heavy and weak. Some ataxia soon presents itself, as is shown by the unstable station with

closed eyes, or difficulty in turning or walking under the same condition. The *bace-jerks* will be found increased in activity and amplitude. The *rotatory reflexes* may be increased or diminished. The *crossaster* is usually inactive. *Muscular tenderness* can be easily detected. The muscles do not lose their contours and are often firm to the touch, but are incapable of strong contraction. The *ataxia* increases; the gait becomes very uncertain, with a tendency to sprawling, and at the same time it is spastic, so that the feet are not readily brought forward, and they may be shaken by *clonus*, which is often easily developed in the calf and rectus femoris muscles. Some *residual weakness* is very commonly encountered, and loss of sexual appetite is frequent, though erection and even painful priapism may annoy the patient.

*Sensory symptoms* are much less common, and when present are extremely slight as compared with tabes. Instead of lightning pains we find dull aches, usually in the thighs and small of the back and over the sacrum. *Parasthetic sensations* are not uncommon, but actual anesthesia or any considerable blunting of tactile sensation is very rare. The *girdle pain* is occasionally present. *Visceral crises* are not common, and are not severe when they do occur. Rectal and vesical *tenderness* and painful *cramps* in the legs at night are sometimes the source of much complaint, and the corresponding *sphincters* may be impaired in exceptional cases. There is often inability to satisfactorily empty the bladder and bowels. Urinary retention may then lead to *cystitis*.

The upper extremities almost invariably show an exaggeration of the tendon reflexes, and some rigidity and weakness is not infrequent. An exaggerated *jaw-jerk* may be easily demonstrated in most instances. Tremulous *twitchings* in the tongue and face are not rare, and speech may be thickened slightly, even in the cases not associated with parietal dementia. *Pupillary symptoms* are not uncommon, but the light-reflex is rarely lost when accumulative contraction persists. Inequalities of the pupils, irregularities of outline, and sluggishness of mobility to various stimuli are frequently noted. The *mental state* is ordinarily normal, but there may be some loss of memory, dependent mainly on lack of concentration and attention. Prolonged mental application is usually impossible.

As the disease progresses the paresis in the legs and the spasticity increase. The static ataxia is engendered actually and also by the increased weakness. Finally, the patient is unable to walk and the spastic ataxia becomes marked in the arms. The paraplegic state is thus induced, but sensory disturbance, as a rule, is insignificant to the last.

**Course.**—While the disease is *progressive* it is much less rapid in its course than tabes, and the great majority of cases never become entirely unable to walk. Of itself it rarely causes death. Buboeris, cystitis, and kidney disease are the chief dangers. Intercurrent affections necessarily find victims in these devitalized subjects. The cases that commence as tabes or as myelitis present features referable to their origin. Early loss of knee-jerks, the presence of girdle pains, anesthesia, analgesia, or joint-lesions with spastic features in the upper spinal levels are probably due to myelitic extension from a posterior sclerosis. Those presenting rapid onset and girdle features and localized atrophy are usually attributable to diffuse myelitis.



**Diagnosis.**—The diagnosis of progressive spastic ataxia, which is the best descriptive name for this symptom group, depends upon the muscular weakness, the ataxia, the spasticity, and the progressiveness of the disease. In early stages when the ataxia is prominent it is usually mistaken for *tubercle*, but the muscular weakness, the increased reflexes, the insignificant sensory disturbance, the absence of girdle pains, and visceral crises should at once differentiate it. In later stages when the paraplegic features are prominent the ataxia is lost in the motor extinction, but a history of it can usually be obtained. Transverse or focal lesions are not sometimes thought of. The sensory integrity and the presence of symptoms in the arms and in the cranial nerves should serve to exclude such limited lesions. The family ataxias present a somewhat similar symptomatology, and the cord-lesion has about the same distribution. The familial features, the nystagmus and articular disturbance, its occurrence in early life, and in the type of Friedreich the absent patellar reflex, should make the diagnosis plain. *Cerebellar tumor* can induce ataxia, increased knee-jerks, and weakness in the legs, but also usually has vomiting, occipital pain, enlarged disc, and retracted head, and may have forced attitudes or movements to distinguish it.

**Prognosis.**—The slowly progressive tendency of the disease has been sufficiently insisted upon. Stationary periods, or those comparatively so, are very common, and even improvement in the individual features of some cases are not infrequent. Complete relief, in the nature of things, is an impossibility, and the onward course of the paretic and spastic features is taken up sooner or later. Mental disturbance, while uncommon, may appear and paretic dementia ensue. Every case is to be judged by itself. The rapidity or slowness of development in the early years of the malady is likely to mark it throughout. As already indicated, only a small portion of the cases reach absolute helplessness.

**Treatment.**—The treatment of a case of progressive spastic ataxia will be modified by its variety and origin. When secondary to *tubercle*, the treatment is the same as in that disease. When *syphilis* is admitted or can be determined by examinations of the blood and spinal fluid, continuous antisyphilitic treatment must be pursued, guided by the blood tests. Counterirritation over the spine, spinal stretching, and exercises to reestablish coordinate motor control are valuable. Thermic baths, and hydrotherapy generally, here find a very useful application. As a rule, the use of hot douches, sprays, and baths lessen the spasticity, but in rare cases cold to the back has the better effect. Massage of the muscles and graduated exercises are important, but the fatigue point should be carefully estimated and never exceeded. To this end the paretic features are the guide, and the sensation of fatigue the criterion. Owing to the hyperexcitability of the reflexes, massage sometimes is not well borne. General measures looking to the physical health and the mental quietude of these patients add to their comfort and to the length of life. The bladder condition should receive constant watchfulness, as a high degree of retention may develop without attracting the patient's notice.

## COMBINED CORD-LESIONS IN ANEMIAS AND CACHEXIAS.

Nissl was the first to demonstrate decided changes of the spinal cord in cases of pernicious anemia. Somewhat similar changes in the retina were well known, and apparently identical degenerations take place in the brain. This observer found degenerative changes in the cords of ten out of seventeen cases of pernicious anemia though symptoms referable to the cord had been observed in but two. The changes are prin-



FIG. 187.



FIG. 188.



FIG. 189.

Figs. 187, 188, and 189.—Sections from three spinal cords showing degenerations due to pernicious anemia (Nissl's).

cipally located in the white matter of the cord, and show a decided tendency to mainly affect the posterior half, giving rise to a group of



symptoms referable to the posterior and lateral tracts. In some instances, however, the anterior portions of the cord and even the gray matter is involved. By experimentally induced anemia in the brain and cord Massaro, Scricchiano, and Soukhanoff have demonstrated similar cellular changes in animals. Tschanneller, Minnich, Liehtheim, in Germany, Russell, Batten, Collier, in England, Dana, Putnam, Riggs, and Billings, in this country, among many others, have not only confirmed Nonne's findings, but broadened the general field of known degenerative conditions secondary to depraved physical states. The consensus of opinion now seems to be that the cord changes are due to a toxic process, secondary to many seriously debilitating systemic conditions, acting upon a nervous system of deficient attributes, shown either by a marked neuritic heredity or by general physical defects.<sup>1</sup> The operation of the toxic process is apparently by way of the vascular apparatus, giving rise to hemorrhagic or interstitial changes resulting in more or less sclerotic transformation.

Women are affected nearly three times as frequently as men.

Symptoms usually appear after the age of thirty, most commonly in the fourth decade of life. In addition to the features of the anemia and cachexia and the conditions arising from these states, there is often a decided mental irritability. Atrophy of the optic nerve has been observed in some instances. Epileptiform attacks have occurred.

The symptoms referable to the spinal cord are those of ataxia, spasticity, and weakness, variously combined, giving rise, as a rule, to an ataxic paraplegia similar to that described in the foregoing section. Early in the disorder the tenderness is to increased reflexes, which later tend to subside or may entirely disappear or persist. In some cases the knee-jerk is lost early. Girdle sensations are not uncommon. Paresthesia occur from the first and are persistent. Neuralgias are common, so that multiple neuritis is often suspected. Bed-sores, cystitis, and sphincteric palsy may be encountered in terminal states exceptionally.

The duration of the disorder is variable. Many cases reach a profound degree of exhaustion before the cord symptoms develop, in which case they are likely to rapidly attain a severe grade. In other instances paresthesia, increased reflexes, and motor weakness come on insidiously and endure for a number of years, the general physical state being relatively but little impaired. In a general way these cases last from one to five years, the tendency being steadily for the worse, though slight remissions may be encountered. In some instances the paralytic and sensory disturbance becomes rather rapidly greater in the lower extremities and steadily mounts upward like an ascending myelitis or may advance and recede many times. The ultimate outcome is almost invariably fatal, and the prognosis depends mainly upon the systemic state.

The treatment is that of the anemia or underlying cachexia, the secondary toxic conditions, and the resulting changes in the cord. In the pernicious types of anemia the use of the newer arsenical preparations, particularly the cacodylate of sodium and neosalvarsan, can be recommended. Normal salt solution by the drop method into the bowel or by hypodermoclysis increases the volume of fluids in the circulation, washes the blood, and favors elimination of toxins. The disolu-

<sup>1</sup>Putnam and Taylor, "Jour. of Ment. and Nerv. Dis.," Jan., 1901.

ties arising from involvement of the cord have to be met by gymnastics, exercises, and massage.

### FAMILY ATAXIA

In 1861 Friedrich reported several cases of ataxia occurring in the children of one family. They presented, among many symptoms, the loss of the patellar reflex, incoordination of all four extremities, rigidity, disturbance of articulation, and a progressive tendency to helplessness. Similar cases have since been reported in every country, until hundreds may be collected from the literature. In some instances the disease is found to appear in several generations, but, ordinarily, it is confined to a single family, and usually appears before adolescence. To the symptom group presented by these cases the name of *Friedrich's disease*, or *Friedrich's ataxia*, has been given.

In 1880 Fraser<sup>1</sup> reported a series of cases, extending through several generations, marked by the same symptoms as occur in Friedrich's group, excepting that the reflexes were increased and ankle-clonus sometimes present. Noone, in 1891, reported a similar family, and clearly pointed out the features in which the cases falling under his observation differed from the Friedrich variety, and especially the fact that they developed later in life,—namely, during or after adolescence,—and that they frequently showed visual defects and optic atrophy. Early examinations of Friedrich's form discovered spinal sclerosis, embracing the posterolateral area throughout the cord, and a cord of diminutive size in some instances. To this, in some cases, is added an undersized cerebellum and cranial-nerve lesions. In one of Noone's cases only an extreme smallness of the cerebellum and cord was found. Switalski<sup>2</sup> reports a case showing diminutive proportions in cord and cerebellum, degenerations of Goll's columns, the direct cerebellar tract, Gowers' tract and some atrophy of the anterior horn cells. The cerebellum showed a diminished number of convolutions, separated by deep fissures, and a paucity of white substance. A family very similar to those of Fraser and Noone was reported by Sanger Brown,<sup>3</sup> and autopsy on three of his cases showed no gross cerebellar defect.<sup>4</sup> Transition cases<sup>5</sup> are being observed that furnish every intermediate variety between the spinal cases on the one hand and the cerebellar cases on the other. For the latter group Marie has used the descriptive designation *pseudosporadic cerebellous*, and *acquired cerebellar ataxia*. Senator<sup>6</sup> sees in Friedrich's ataxia only the manifestations of teratological cerebellar and spinal defect. In the cases of Noone and others marked by cerebellar atrophy, or, more probably, cerebellar agenesis, the same origin is apparent, and the variety of clinical cases and anatomical findings seems to depend upon the location of the principal defect. Gordon Holmes in a careful review of the subject,<sup>7</sup> basing his groups upon definite and carefully investigated cases which have been properly examined post mortem, suggests the following classification: (1) Primary parenchymatous degeneration of the cerebellum. (2) Olivo-ponto-cerebellar atrophy. (3) Degeneration of the spinocerebellar tracts, the cerebellum being normal or small only. (4) Congenital smallness of the central nervous system with cerebellar

<sup>1</sup> "Glasgow Med. Jour.," 1880.

<sup>2</sup> "Nouv. Icon. de la Salpêtr.," Sept., 1904.

<sup>3</sup> "Brain," 1902.

<sup>4</sup> "Ibid.," 1897.

<sup>5</sup> Henshaw, "Rev. de Méd.," 1895. <sup>6</sup> "Berlin klin. Wochens.," 1903.

<sup>7</sup> "Brain," No. 120, 1907.



symptoms. The familial and hereditary features of these cases at once declare their embryonic character and origin. The portions of the cord that undergo sclerotic changes are precisely those which are last to develop and become myelinated, only reaching completion at the end of the ninth or in the tenth month.

**Etiology.**—The most important etiological feature of the family ataxias is their embryological nature. Why a mother should give birth to several children with defective nervous structures or other teratological defects it is impossible to say, though phthisis and other exhausting endemic have presented in some of these parents. As is common in familial nervous diseases, there is a tendency to a preponderance of males, and the transmission is usually by the female line. Both of these points are illustrated in the genealogical diagram of Brown's cases (Fig. 200). In a given family there is frequently noted a tendency for the disease to be manifest at a progressively earlier age in successive children. The symptoms are likely to appear at the developmental periods of life. In some cases they are congenitally evident; in others the first or second dentition, the ages of puberty, adolescence, and complete adult sexual differentiation and reproductive ability seem to make demands that the defective neural apparatus can not meet, and thereafter retrogrades. In exceptional cases the disease appears late in life. Friedreich's type usually occurs before the age of fourteen; hereditary cerebellar ataxia, or Marie's form, after pubescence. In many cases, especially in children, and, therefore, usually in Friedreich's variety, the

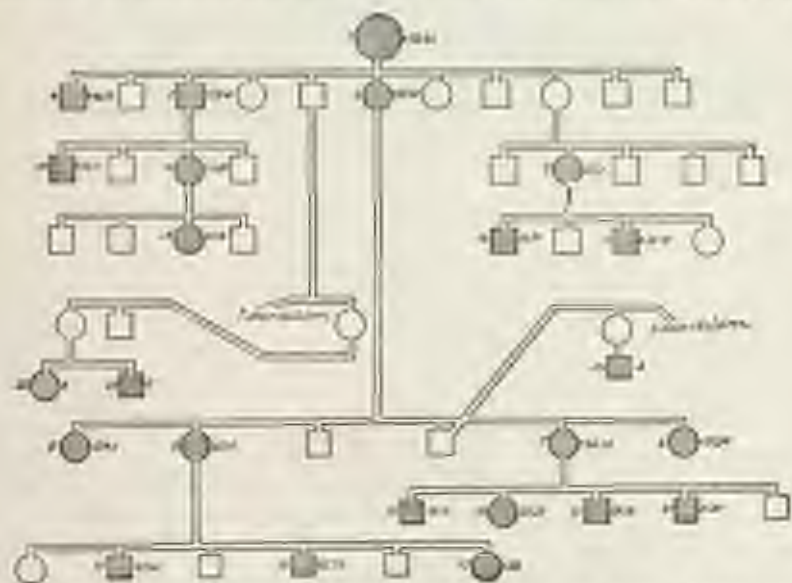


Fig. 200.—Family tree of hereditary ataxia reported by Dr. James Brown. Explanation of diagram: Shaded symbols indicate hereditary ataxia. Arrows indicate males; circles females. The numbers in the left side to the cases in Brown's paper; the first number to the right, the age at death at the present age; † indicates deceased. The last number indicates the age at onset.

occurrence of an infectious fever appears to precipitate the symptoms of the disease. In these cases, too, the cord lesions correspond to those of the combined sclerosis and occupy the posterior arterial field of the cord's cross-section. The embryonic vulnerability of this portion of the cord may

serve to locate the lesions in these instances in the lower levels through the intermediary of the vascular supply and the action of toxic factors.

**Morbid Anatomy.**—The morbid anatomy of these family ataxias varies as the case corresponds to the spinal or the cerebellar type. The cases that conform strictly to *Friedreich's syndrome* show a posterolateral sclerosis analogous in distribution to that of progressive spastic ataxia. There is a decided sclerosis of the columns of *Gall and Burdach*, with shrinking of the cord in this region throughout its entire length. There is sclerosis of the crossed pyramidal tract, of *Gowers' tract*, of the direct cerebellar tract, of *Lissauer's tract*, and frequently atrophy of the cells of *Clarke's column*. The posterior horns and its cells are shrunken and in rare cases the anterior cornual cells are degenerated. Marie insists that the changes in the pyramidal tract are confined to the fibers related to the direct cerebellar tracts and *Gowers' tracts*, and do not directly affect the upper motor neurons. The portions of the cord that last develop are thus affected by the retrogressive changes of the malady. As a rule, the cord is undersized and may present only two-thirds of its usual thickness. In a few cases, notably that of Menod, the medulla and cerebellum were also reduced in size and degeneration has been traced into the cerebellum. The posterior roots and ganglia are usually normal.



Fig. 201.—Coronal section in *Friedreich's disease*, showing distribution and relative intensity of atrophic changes (Marie).

Rennie,<sup>1</sup> however, found degeneration of posterior root-fibers, and posterior root-zones. The cranial nerves are generally spared in the spinal variety, but the hypoglossus and optic nerves have been found diseased.

The cerebellar form, in the few cases on record, has presented atrophy of the cerebellum, as in *Fraser's case*, in one of *Neume's*, in *Saitalskii's*, and in *Holmes' case*.<sup>2</sup> This is absent in *Brown's cases*, but the cerebellar tracts in the cord were involved. This cerebellar atrophy is said not to be sclerotic. The gray substance is abnormally thin, *Purkinje's cells* are few in number and undersized, the white matter is less voluminous than usual, and the weight of the organ is reduced a third or a half. The cord in these cases of *Marie's type* is undersized, but shows no sclerotic degenerations. There appears to be a pure genetic poverty of fibers, or else they have disappeared, leaving no trace. In *Brown's cases*, as reported by *Meyer* and later by *Barker*,<sup>3</sup> there was degeneration in the dorsolateral cerebellar tract and degeneration in the gray and white matter of the cord, medulla, and cerebellum. Optic atrophy is not infrequent.

<sup>1</sup> *Br. Med. Jour.*, July 15, 1890.

<sup>2</sup> *Brain*, 1907, No. 120.

<sup>3</sup> *Dissect. Pub.*, University of Chicago, 1900.



**Symptoms.**—The *motor symptoms* are the most prominent. They commence with clumsiness, especially in walking. The child stumbles over every trifle and becomes more and more unsteady on its feet. Later the gait is staggering and drunken in its uncertainty. Ataxic marks every movement and position, but there is no rigidity even in the cases presenting increased reflexes. All movements are clearly intentional, though clumsily executed, and leave for their purpose to maintain the insecure equilibrium. In standing the body is constantly swaying, and shuffling steps in various directions are frequently taken to maintain the balance. With closed eyes the difficulties of the station and gait are not totally increased, and there is practically no loss of the *muscular sense*, only an ataxic incoordination in executing movements. Frequently coarse, rhythmic, *trembling* or *jerky movements* are made on attempts to use the extremities, and at rest often a finger or the wrist, shoulder, elbow, head, or lower extremity is moved or twitched in a spasmodic manner. Such movements are frequently seen in the face, especially when the patient begins to talk or when some emotional expression is called out. Oculum, complete support of the part



FIG. 202.—Ataxic gait in Friedreich's ataxia. 1, G. F., eight years old, affected one year; 2, P. F., thirteen years old, affected three years; 3, M. T., twenty years old, affected five years.

at its complete rest causes them to cease. The head, in a similar way, nods in one direction or another, and may, in late cases, roll around on the shoulders as if articulated with ball and socket. As the patient sits, the body may also sway about above the hips. An examination of the *muscular strength* finds it but slightly reduced, and clearly indicates that there is no serious paralytic state present. The muscles do not waste except in cases of long standing, and then only rarely and mainly about the shoulders and hands.

The *sensory symptoms* have usually been given little attention because the superficial elements of touch are not much modified. A careful study of sensation in 20 cases by P. W. Saunders<sup>1</sup> brings out the important fact that the elements of sensibility which are supposed to be conducted through the dorsal columns of the cord are constantly disturbed, and more especially for the lower extremities. Touch, pain, and temperature tests are not much modified, though usually reduced to some degree, but the recognition of vibrations, the sense of passive movements and position, and the appreciation of double contacts are greatly diminished.

The *superficial reflexes* are not disturbed. In cases of the Friedreich

<sup>1</sup>"Brain," 1913, p. 106.

type the deep reflexes are diminished or abolished and the knee-jerk usually disappears very early. In Marie's form the deep reflexes are exaggerated and foot- and rectus-clonus are commonly encountered. The *spinifers* are unaffected.

The *face* presents an appearance of listlessness that deepens as the disease advances and in some instances first calls attention to its invasion. The features droop in a mask-like blankness of expression, often intensified by a half-open mouth. In emotional expression the ataxic unbalance appears, resulting in convulsions, or, at least, exaggerations of facial movements. There is usually well-marked *nystagmus*. The oscillations of the globe, however, tend to cease if the eye is allowed to remain at rest, and vary in amplitude and are increased by effort, like all the other manifestations of incoördinate muscular balance. It is often necessary to direct the eyes widely from the direct line of forward vision to develop the *nystagmic jerking*. This can usually be accomplished by having the patient fix his eyes upon an object in an outward and upward direction. Pulsos of the ocular muscles are extremely rare. Jeffroy has published one case, and another is illustrated here. In Friedreich's form *optic atrophy* is a rare exception. Strahl<sup>2</sup> reports three out of four cases in one family showing some degree of it. In Marie's form, on the other hand, it is a common finding, and changes in the visual acuity and in the form- and color-fields are frequently encountered. The *papillary reflex* is usually normal in Friedreich's form, but may be affected in Marie's group.

The *speech* is ataxic, if the expression may be used. *Modulation* of the voice and prompt *consonation* are defeated by the tardy and incoördinate action of the muscular apparatus. It reminds one of the gait; some words come quickly, others slowly, and the voice tones change suddenly and irregularly in pitch or other qualities, though tend-

ing to a monotone. The *intelligence* of these patients is not much impaired in the early years of their malady, and is often belied by their apathetic and empty faces, but in the later stages of the disease the mind is frequently sluggish or shows some retardation of development. In Friedreich's form physical development is also frequently retarded if the disease manifests itself before puberty, and sexual functions are exorbitantly backward.

*Trophic and vasomotor defects* are very rarely encountered, or are entirely lacking except a peculiar conformation of the feet and a slight vertebral scoliois that are frequent in Friedreich's form. The *club-foot* consists of an exaggeration of the plantar arch which shortens the foot notably and causes the toes to assume the



FIG. 30.—Clubfoot of Friedreich's disease, showing shortened arch and increased gait toe.

"hammer" position, extended at the first and flexed at the other phalan-

<sup>2</sup> "Med. Rec.," N. Y., July 20, 1893.



ges. This is particularly marked in the big toe, which usually is first affected and is sometimes the first symptom of the disease detected by relatives conversant with its forms. The deformity is increased when the foot is extended upon the leg, and if slightly marked, may disappear when the patient is standing. A similar deformity of the feet is also encountered in some of the family and sporadic cases of progressive muscular atrophy. The *scoldosis* appears in most of the well-developed cases of Friedreich's form, and occupies the dorsal and lumbar spine. It is usually slight.

**Varieties.**—The two major varieties making up the group of family ataxia have been indicated throughout the preceding pages, and there are many authoritative writers who regard them as distinct entities. Their principal differences are contained in the following table. The cerebellar features clinically predominate in both and ataxias are yet too few to justify their complete separation, especially as all intermediate varieties are being reported:

FRIEDREICH'S FORM.	MARSH'S FORM.
Hereditary spinal ataxia appears usually before puberty.	Hereditary cerebellar ataxia appears usually after puberty.
Clonic tremor in upper extremities and oscillation in head and trunk frequent.	Very common and pronounced.
Optic atrophy and amblyopia very exceptional.	Common.
Tendon reflexes diminished or absent. Club-foot and scoliosis common.	Increased, foot-clonus frequent. Exceptional.

**Course.**—The first symptoms to attract attention, unless the case is congenital, are the difficulties in walking. Sometimes certain families, who have learned to know the symptoms from the number of cases among them, recognize, by the *facies* or the *scoldosis* or the club-foot and cramped toes, that another member has been added to the list. In the course of three to four years the ataxia in all four extremities is well marked. It advances and augments and in another similar period takes the patient off his feet and renders him practically helpless. In this state he may live ten to twenty years and finally die from intercurrent disease. While the course of the disease is commonly steadily progressive, long remissions may occur and sudden aggravations take place, but death does not result directly from the disorder.

**Diagnosis.**—The diagnosis of a case of family ataxia is almost self-evident if numerous members of the same or succeeding generations are affected, but in certain instances no such collateral cases exist. The differential diagnosis must then be made from *tubercles*, *chorea*, and *insular sclerosis*. In *tubercles* the gait lacks the cerebellar quality. Posterior sclerosis also presents visceral crises, lightning pains, vesical weakness, sensory disturbances, and pupillary symptoms. It is a disease of full adult life, has commonly a syphilitic history, and nystagmus is extremely uncommon in it. The cases of family ataxia occurring late in life usually present increased reflexes.

From *chorea*, for which it is frequently mistaken, especially in children, family ataxia may be distinguished by the major affection of the upper extremities in Sydenham's disease, its rather abrupt onset, and

the absence of nystagmus, sciosis, clubfoot, and persistent abnormalities of the tendon reflexes. Huntington's family chorea of adult life has its mental features, post forced attitudes and movements, which are greatly in excess of anything seen in the family ataxia.

*Jacobian sclerosis* presents the closest resemblance to family ataxia, especially Marie's form; and as it also may affect several members of the same family, the differentiation may be most difficult, if not impossible. We must recall its distinctive intention tremor, its lack of static instability, its scanning staccato speech, its spastic features and gait.

**Prognosis.**—The outlook in these family ataxias is always gloomy. Aside from the helplessness there is no suffering, and, as a rule, they bear their lot with an apathetic good nature.

**Treatment.**—The management of these cases does not promise much improvement. Some have thought that spinal stretching and electricity improved the incoordination. Carefully planned exercises may, perhaps, assist in the same direction.

### HEREDITARY SPASTIC PARAPLEGIA.

Commencing with Strümpell, groups of cases have been recorded by Bernhardt, Phillips, South, Newmark,<sup>1</sup> Achard and Frossen,<sup>2</sup> Bayl,<sup>3</sup> and others, which present pure spasticity and familial traits. Bayl traced the disease through five generations, the individuals affected showing marked similarity in all respects. In his series it seemed that the disease once escaped did not reappear in the descendants. Spiller<sup>4</sup> reports a family showing the disease in eight generations.

The spastic condition may appear at any age from the first (Achard and Frossen) to the fifty-sixth year (Strümpell) and very commonly is preceded by some infectious disease, as typhoid, measles, etc. There is paresis only in the terminal stages and no ataxia. The muscular hyperreflexus and increased reflexes are alone noted. Sensory and sphincteric difficulties are absent and cerebral features are insignificant, as only such as are attributable to defects in the intracerebral portion of the motor pathway. The leg rigidity may, however, render the gait markedly spastic and walking finally impossible.

The **symptoms** are referable to the pyramidal tracts which, with the columns of Goll and the direct cerebellar tracts, were found degenerated by Strümpell. Erb,<sup>5</sup> who called it a primary spastic paraplegia, has collected ten postmortem reports showing the sclerosis to be mainly confined to the lateral portions of the cord and principally to the crossed pyramidal tract. Jendrassak<sup>6</sup> believes that the process may begin in the brain, bulb, or cord, and is intrusive in nature. From the familial feature of these cases it is evident that the basis of the paraplegia is a hereditary defect and its sometimes late appearance is no bar to such belief.

**Treatment** promises little, though massage and carefully selected exercises may accomplish something.

<sup>1</sup> *Med. News*, Feb. 18, 1897.

<sup>2</sup> *Quint. Journ. de Med. et de Chir.*, Dec., 1896.

<sup>3</sup> *Jour. Nerv. and Ment. Dis.*, Nov., 1897.

<sup>4</sup> *Wash. Med. Jour.*, June 21, 1902.

<sup>5</sup> *Br. Med. Jour.*, Oct. 11, 1902.

<sup>6</sup> *Deut. Arch. f. Klin. Med.*, Bd. 144.



The prognosis is not hopeful, but the course of the disease is usually very slow and for a given family nearly the same in all the affected members.

#### SCLEROSIS OF THE CORD DUE TO VEGETABLE INTOXICANTS— ERGOTISM, LATHYRISM, PELLAGRA.

**Ergotism.**—Epidemics of ergotism have arisen in various localities of the Old World, usually in times of bad harvest, when the people were reduced to eating ergoted grain. Brain symptoms in the form of mania are often associated, and epileptoid attacks are common. The disease respects neither age nor sex, and sometimes animals present similar cord symptoms. These, in man, consist of paresthesias, lightning pains, girde sensations, analgesia, static instability, and an ataxic gait with obliteration of the patellar reflexes. Neither optic atrophy nor pupillary stasis occurs. In all recent autopsies a sclerosis confined to the posterior columns, and conforming closely to the changes in tubes, has been found. In many instances the symptoms develop after the poisonous food has been discontinued for some months. The tendency of the disease is toward recovery by progressive amelioration during several years. In some instances even the knee-jerk is restored. The mortality, however, in acute cases, is rather great. (For fuller particulars the reader is referred to the article of Tuzsek, "Archiv f. Psychiatrie," Bd. xiii, 8, 99.)

**Lathyrism.**<sup>2</sup>—Epidemics of this disease in Europe, Africa, and Asia have been noted for centuries. The cases are marked by rigidity of the lower extremities coming on within a few days, and paraplegic symptoms, with weakness of the bladder, generally ending in slow recovery. They coincide with famine conditions, when the people eke out their subsistence with the various vetches, such as *Lathyrus sativus*, or *avena*, or *elymus*. The noxious constituent appears to be an alkaloid not destroyed by ordinary cooking. The symptoms come on gradually or sometimes rather abruptly, and a spasmodic paraplegia is developed, producing the paraplegic state, so far as motion and locomotion is concerned. Sensation is not materially disturbed, though paresthesias and some blunting of cutaneous sensation are usual. Mental symptoms and cerebral features, including involvement of cranial nerves, seem to be lacking. The exact lesion is not known, but the symptoms all point to disturbance in the lateral tracts, or, perhaps, to a posterolateral sclerosis with preponderating spasticity. Often such cases suggest a focal lesion of the cord, but the absence of serious sensory disturbances and the complete recovery that, as a rule, ensues are not consistent with such a point of view.

**Pellagra**, from two Italian words meaning rough skin, is also known as pellarelli, Alpine scurvy, Asturian leprosy, dermatagra, etc. Formerly it was mainly of European interest, being encountered principally in southern Europe, especially in northern Italy, and, to some extent, in northern Africa. It has been observed in Mexico, South America, and the West Indies. Although cases were reported in this

<sup>2</sup> Brunsell, "Trans. Seventh Internat. Cong.," vol. 9, p. 45.

country by Gray, of Utica, N. Y., in 1864, only since 1907 has its wide distribution in the United States been recognized. Lavinder<sup>1</sup> estimated that there were 1500 cases in the southern States between 1906 and 1909. Several hundred cases were found in Illinois institutions for the insane in 1909, and scattered cases have been reported in many States. Pellagra may be defined or described as a periodic and progressive, non-contagious, non-inheritable disease, of insidious course, characterized by a peculiar, periodic eruption, and a series of symptoms involving the nervous and digestive systems (Roberts).

Lombroso, among the early Italian authors, so emphatically attributed pellagra to maize, and particularly to bad or spoiled maize, that his views have always strongly colored the view. There is no positive evidence that Indian corn or its products plays any direct part in the causation of pellagra. Sambor<sup>2</sup> denies the corn etiology altogether, and calls attention to the well-defined topographical limitation of pellagra in Italy to valleys and streams infested by a certain fly (*muscidus*), which he thought served to explain the seasonal variations of the disease. Roberts<sup>3</sup> finds similar conditions to obtain in the State of Georgia, but a particular parasite is not yet in evidence. Alessandrini and Sciala<sup>4</sup> also deny the influence of maize, but attribute the disorder to colloidal solutions of silica derived from clay soil and carried in the drinking-water. Goldberger<sup>5</sup> claims that the disease is purely nutritional and reports three widely separated groups of cases most of which were cured by an improved dietary. Moreover, by limiting the diet in a group of convicts who volunteered for the purpose he produced pellagra in six out of eleven cases. Pellagra must undoubtedly be considered as one of the deprivation order due mainly to an absence or paucity of albuminoid food constituents. Its prevalence in Serbia and Roumania in the present war is quite in accord with the definite observations of American authorities.

Conditions of impoverishment and exposure to the sun are such common factors as to be almost essential. The disease has a distinct relation to the summer season, and shows a strong tendency to recurrent annual attacks. Males and females are commonly said to be equally affected, but R. M. Grunier,<sup>6</sup> in a survey of twenty-five counties in Kentucky, South Carolina, and Georgia, embracing 1426 cases, found females more than twice the number of males, and whites much outnumbered the blacks.

The symptoms of pellagra are of four orders, mental, cutaneous, intestinal, and nervous. Biondini says that in Italy the disease appears in the spring, but more commonly in May and June, with general weariness and depression. Relapses occur at the same annual periods. Pains, parasthesias, vertigos, and malaise are soon associated with a dark

<sup>1</sup>"Pub. Health Report," 1909.

<sup>2</sup>"*Ann. of Tropical Med.*," London, 1910.

<sup>3</sup>"*Amer. Amer. Med. Assoc.*," June 11, 1911.

<sup>4</sup>"*Rivista*," 1914.

<sup>5</sup>"U. S. Pub. Health Reports," Oct., Nov., 1915.

<sup>6</sup>"Pub. Health Report," March, 1913.



erythema of symmetrical distribution on the portions of the body exposed to the sun—the neck, hands, and feet. It is sharply demarcated from the adjacent healthy skin, and its outline is dependent upon the protection afforded by clothing; even rings, wristbands, and similar articles protect the underlying parts. When the erythema subsides, the skin remains larch, thickened, wrinkled, and very much changed. Itching is pronounced. Blisters and bullæ sometimes appear, a moist retentive appearance is more common, and small scales form and fall rapidly. A general dyscrasia develops; there are stomatitis, lack of appetite, gastric and abdominal pains, and persistent diarrhea. With advancing prostration, dejection and melancholia appear. Parosæsthesias, cramps, ataxia, increased reflexes, and motor weakness are observed in a majority of cases. In a lesser number there are excitability and maniacal phases, and in a few some of the mental and physical peculiarities of general paresis, such as expansiveness, egotism, excitability, with ataxia and increased deep reflexes.

The American cases present some variations from the foreign types in that the erythema is usually not presented until the second year, and shows a greater intensity. Conl symptoms are less pronounced, and conl changes are less frequently mentioned in the autopsical records.

The conl-lesions<sup>1</sup> that are found consist of a leptomeningitis, often with much thickening, and even with the formation of osseous plaques. In the conl itself the anterior conal cells are frequently atrophied and pigmented. There is commonly a posterolateral sclerosis. This affects the columns of Goll and Burdach, mainly in the upper conl-levels, but spares the root-zone of the postero-external column. The crossed pyramidal tract, especially the lower portion, is also sharply sclerosed, the direct cerebellar tract usually escaping.

The spinal symptoms correspond. Ataxia is most marked in the upper extremities; spasticity is pronounced in the lower limbs. The



FIG. 25. a.—Pellagrous erythema of the hands with gross thickening of the skin (from the Bulletin of the Illinois State Board of Health, August, 1905).

Indian reflexes are spared, and cutaneous sensibility is not much affected. Strangely, in spite of the usual changes in the anterior conal cells,

<sup>1</sup> Tassin, "Meningo-encephalitis Pellagrosa," 1892.

vascular atrophy is insignificant. The disease, clinically and anatomically, sometimes presents much resemblance to parietic dementia.

The course of the disease varies to such a degree that acute and chronic cases are everywhere encountered, the first frequently terminating fatally in a few months, the latter extending over many years with annual recrudescences. The tendency is to chronic insanity, especially melancholia and terminal dementia, with a maniacal fatal exit, although recoveries are not uncommon.

In the treatment serums and drugs have thus far equally failed. Reliance is mainly to be placed upon a properly balanced diet, under which many cases make prompt improvement and permanent recovery.<sup>1</sup>

<sup>1</sup>Goldberger, "Jour. Amer. Med. Assoc.," Feb. 12, 1916.



## PART VI.

### DISEASES OF THE GENERAL NERVOUS SYSTEM WITH KNOWN ANATOMICAL BASIS.

AMONG the various diseases that have already been described, several have presented symptoms referable to the various portions of the entire nervous apparatus and properly belong to this section. Thus, in *multiple sclerosis* all nervous levels may be invaded. Cerebral, spinal, and nerve-trunk lesions are all encountered. The same is true of *cerebrospinal meningitis*, *tubercle*, *paratyphoid fever*, *tuberculous meningeal atrophy*, *erythema*, *leptospirosis*, *poliomyelitis*, and *polioencephalomyelitis*; but for purposes of comparison as well as contrast it was thought most expedient to take them up in the order chosen, being guided by the preponderance of nerve-trunk or spinal-cord features in each instance. To these must be here add *multiple hereditary sclerosis* and *sypilis of the nervous system*. With some of the syphilitic lesions we are already acquainted.

## CHAPTER I.

### MULTIPLE CEREBROSPINAL SCLEROSIS.

*Multiple cerebrospinal sclerosis—multiple sclerosis, insular sclerosis, sclerosis en plaques, or disseminated sclerosis*—is a disease secondary usually to some intoxication or infection often of a mixed sort. It is marked by numerous islets or plaques of sclerosis irregularly distributed in the brain, cord, and cranial nerves. These are related to the blood-supply of the part and probably primarily vascular.

**Etiology.**—This disease is not very common. For instance, Uhthoff in six or seven years could only gather about 100 cases from all the hospitals and clinics of Berlin. It affects both sexes indifferently and presents a large preponderance of cases between the ages of twenty and thirty years. Marie asserts its absolute rarity after forty, but cases do occur. Children are sometimes affected and it may even be congenital. Considerable stress was formerly laid upon heredity, but this element is chiefly manifest in a neuropathic tendency. Erb, Oppenheim, and Duchenne have cited examples of direct trans-

sion, and it has, in a small number of instances, affected several children of the same family. Herveyet has even seen nine cases in one generation. *Oreocory, scab, transudata*, and various *crocus* have been accused as causative. Such relation is subject to doubt, though all these influences are capable of aggravating the disease when once established and of precipitating additional manifestations of its activity. Hoffman,<sup>2</sup> for instance, in a study of 100 cases, attributed the cause to trauma in 13, but in one-half of the cases no alleged cause could be mentioned. The most important etiological factors are the *infectious*. Typhoid, pneumonia, malaria, measles, scarlet fever, small-pox, diphtheria, whooping-cough, erysipelas, dysentery, cholera, influenza, and the puerperium have been followed by the development of the symp-



Fig. 264.—Section of cerebral substance in the brain (Charnet).



Fig. 265.—Section of the brain substance in the pons and medulla (Charnet).

toms of multiple sclerosis within a few weeks or months.<sup>2</sup> Oppenheim<sup>3</sup> found that in eleven out of twenty-eight cases treated by him the patients had long been exposed to the influence of lead, copper, zinc, etc.; and emphasizes the significance of occupations attended by such intoxications. Charnet, in one of his later lectures, reported a case consecutive to cerebral rheumatism. Infection may explain some of the family groups of multiple sclerosis and Oppenheim's observation of the disease in a mother and child. From the numerous and often mixed infections noted, it seems unlikely that we have to deal with a specific organism. It is also a debated question whether the malleol microbes act through their elaborated poisons or by embolic colonization. It is, however, quite definitely settled that the irritative influence reaches the central nervous organs through the blood-channels and there sets up the localized sclerotic processes. Serodiagnosis and spinal fluid examinations have indicated a syphilitic factor in a considerable num-

<sup>2</sup> "Deut. Zeit. f. Nervenheilk.," Dec., 1901.

<sup>3</sup> Williamson, "Bris." 1896.

<sup>4</sup> "Berlin Klin. Wochens.," March, 1896.



ber of cases (Neuner). Such cases must be considered either as multiple sclerosis plus syphilis or as discrete syphilitic lesions of the nervous system,<sup>1</sup> or that syphilis is the agent provocateur of the gliosis (Spiller). Early sclerotic patches ordinarily show a centrally diseased blood-vessel, and thrombi have been found in several instances which Ribbert,<sup>2</sup> with Marie, is disposed to attribute to microbial emboli.

Goldscheider<sup>3</sup> declares the process analogous to that of an acute myelitis originating through the vascular supply, while Schuster and Biechowsky<sup>4</sup> consider the change as primarily interstitial.

**Morbid Anatomy.**—*Macroscopically* the meninges retain their normal appearance. The pia mater is translucent and frequently the sclerotic patches on the cerebral and cord surfaces can be seen through it. These present various sizes and shapes and are usually grayish or pinkish gray in color. In some cases they may only appear after undergoing the action of bichromate and other hardening solutions. In distribution they recognize no law. Sometimes they are most frequent on the surface; sometimes they are confined to the interior of the cord and brain. In a general way they seem to prefer the white substance to the gray matter. They are found from the cerebral convolutions to the filum terminale, and in variable numbers, from a few to several hundred. Their dimensions are those of a millet-seed to a walnut or larger, and they are often of irregular, but almost invariably of definite, outline. They may invade the cranial nerves, especially the optic, and the spinal roots are not exempt. On section, they present a retracted appearance if old, a salient aspect if recent. Both sorts may be found in the same case.

*Microscopically* the definite outline of the plaques is still maintained, and they are sharply distinguished from the surrounding normal tissue. The axoplasm has disappeared from the nerve-fibers which pass through them, but the axis-cylinders usually persist and sometimes are enlarged. In recent plaques there is an abundance of granular bodies throughout the islet, but in older lesions they are confined to the periphery of the plaque. Through and everywhere in the plaque is a proliferation of the neuroglia, at once the evidence and the result of the irritant cause of the lesion. Observers are agreed that secondary degenerations do not,



Fig. 26.—Distribution of lesions of multiple sclerosis in the cord. Sections taken at various levels throughout the low lumbago (thoracic).

<sup>1</sup>Head and Farnside, "Brain," 1914, vol. xxvii, p. 1.    <sup>2</sup>"Virchow's Archiv," 1905.  
<sup>3</sup>"Zeit. f. klin. Med.," 84, 303.    <sup>4</sup>"Neurolog. Centralbl.," 1907.

as a rule, follow the development of these sclerotic patches. Deprived of their myelin covering, insulation is probably impaired in the nerve-fibers, and to this fact some writers attribute in part the trembling and other motor disturbances commonly presented. Occasionally myelinated fibers are found in old patches, and it has been suggested that they may be due to a regenerative process. These particularly occur in the pyramidal tracts and in Goll's columns. Popoff insisted that the alleged neuroglial hyperplasia does not exist, but regards this appearance as due to degeneration products of the myelin and axis-cylinders. Redlich,<sup>1</sup> however, in a later study, insists fully upon the glial increase in all cases, and Spiller<sup>2</sup> is disposed to designate the plaques as multiple gliomata, a view which is sustained by Kingston.<sup>3</sup> The nerve-cells embraced in the plaques are diminished in size, often pigmented, and their processes are atrophic or may have disappeared.

In the optic nerve there is the same interstitial increase without division of axis-cylinders, and, according to Uhloff, papillitis and atrophy of the disc only occur when the plaque is situated immediately behind the globe.

The vessels in the plaques show decided changes. The coats are thickened, especially the external tunic, so that in section they remain open and dilated. The perivascular sheaths are also dilated and at times obliterated. In other instances thrombotic occlusion of the vessels has been observed. One vessel particularly altered is usually found near the center of each small sclerotic patch, and the sclerosis is most intense at and about this point. The appearance indicates that an early embolism or thrombosis sets up vascular lesions extending to the perivascular spaces, and entails irritative sclerosis of the adjacent neuroglia.

A certain number of cases must be kept in mind where the early changes are due to diffuse myelitis or cerebellitis, or both, and in which secondary degenerations and numerous localized sufferings give rise to a similar clinical picture.

**Symptoms.**—Wide-spread as are the lesions and haphazard as is their location in multiple sclerosis, the clinical symptom group of the fully developed disease is tolerably uniform and ordinarily easily recognized.

The motor features constitute the most important group of symptoms in multiple sclerosis. The gait is usually disturbed at an early period of the disease. Sometimes it is clearly and purely spastic, identically the same as that presented in the paraplegic state: legs rigid, knees abducted, toes turned in and dragging along the ground; clonus showing in the trembling, shaking legs, which are pulled up and advanced by swaying movements of the body and pelvis; all reflexes exaggerated. To this is usually added more or less uncertainty of equilibrium, and we have a combination of cerebellar staggering and spastic rigidity which has been called the *cerebellospasmodic* gait, and is the one most frequently encountered. In exceptional cases the gait is purely cerebellar in quality, without any rigidity, and with diminished or normal reflexes. There is commonly more or less muscular weakness, which may reach a paraplegic degree and defeat all attempts at

<sup>1</sup> *Neurolog. Centralbl.*, 7, 1896, p. 562.

<sup>2</sup> *Arch. of Neurology*, vol. i, p. 219, February, 1919.

<sup>3</sup> *Ibid.*, 145.



walking. One leg is often much more affected than the other, and this may even alternate from side to side at different periods of the disease. In exceptional instances the lower extremities are not affected. Exaggerated deep reflexes are the rule, and the Babinski is a most valuable sign in many cases that show no other indication of organic disease. On the other hand, the abdominal reflexes are very commonly defective and frequently entirely abolished on either one or both sides.

A considerable proportion of cases present apoplecticiform attacks, followed by transitory paralysis at some period of the disease. In rare instances such an apoplecticiform attack is the initial manifestation of the disease.<sup>1</sup> Sometimes both limbs and the face on the same side are involved; sometimes we have a crossed paralysis; sometimes the face escapes, and sometimes the palsy is paraplegic in distribution.

The upper limbs present not only the spastic weakness already noted in the lower extremities, but here is developed to its highest degree the so-called *intention tremor*, which is so characteristic of the disease. While at rest the hands, trunk, and head are perfectly quiet, but once the patient attempts to reach any article, especially if the movement be somewhat extensive and requires precision, the entire member becomes animated with a coarse trembling which augments in amplitude as the movement continues. Finally the object is seized with considerable rudeness. The moment the arm is again placed at rest, and supported so that no muscular effort is expended, the tremor at once

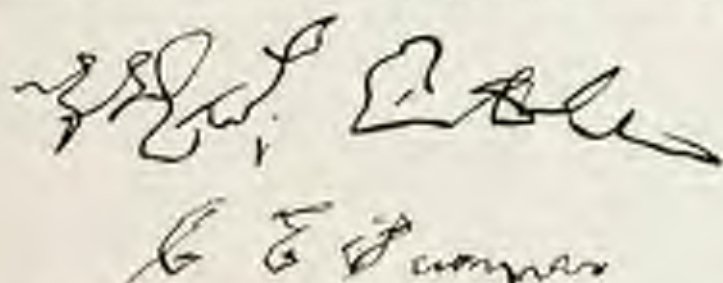


Fig. 26.—Handwriting in two cases of multiple sclerosis, showing intention tremor.

ceases. This tremor shows clearly in the handwriting of these patients, and writing may finally become impossible. Unlike the trembling of paralysis agitans, which is most pronounced in the fingers and while at rest, the intention tremor of multiple sclerosis usually arises at the roots of the extremities, and they are moved in their entirety from the shoulder- or hip-joints. This accounts for the wide range of the tremor excursions, and perhaps for their slow rate, which usually is between five and eight to the second. The lower extremities may also present this intention tremor to some degree, and it frequently shows in the *trunk* and *head* of the patient if without back-support or head-rest. The movements of the head are usually anteroposterior in direction, but may also be somewhat lateral and then tend to produce circumduction of the head upon the shoulders. Frequently the tremor is much more marked on one side than on the other, and rarely it is unilateral.

<sup>1</sup> Boulogne, "Rev. de Méd." May, 1880.

One of the ordinary tests of this tremor is to direct the patient to take a drink from a tumbler of water. In marked cases the tremor appears directly the hand starts toward the glass. This is reached in a fairly direct line, grasped suddenly, and with increasing oscillations carried toward the lips, which are protruded to meet it, with the head and body advanced and shaking. The motions become more and more vigorous, and finally the glass is rattled or dashed against the teeth, and the water not already spilled may drench the patient's face and clothing. The little remaining is obtained by steadying one hand with the other, and firmly holding the glass with the mouth. This tremor is increased by *co-terminating motions*, and is somewhat proportionate to the extent and duration of the movement attempted.

Sensory features in multiple sclerosis, in a general fashion, may be said to be insignificant, so far as *general sensibility* is concerned. Occasionally a patient complains of anomalous sensations of heat or cold, formication, fullness in the limbs, and sometimes of pains which may even be lightning-like in character or of the girdling form. According to Freund, the slight objective sensory changes sometimes encountered are confined to the fingers and toes mainly, and to the distal portions of the limbs. They consist of disturbance of touch, of hyperalgesia and analgesia, and insignificant modifications of thermal sensibility. They are often transient and changeable. F. von Goltz<sup>1</sup> claims that in advanced cases certain areas present permanently disordered sensation. Exceptionally very distinct disturbance of sensation of segmental distribution similar to that of a Brown-Séquard paralysis or a transverse cord lesion may be encountered. I have seen such a one persist for several years. Hemianesthesia and other hysterical distributions of anesthesia sometimes complicate multiple sclerosis, as the diseases may be, and often are, associated.

*Hearing, taste, and smell* are singly disordered in rare cases of multiple sclerosis, usually in the direction of deficit, and may be completely lost.

Disturbance of sight, on the other hand, is frequent, often very marked, and of great diagnostic value. The ocular apparatus may be affected in any portion of its neuromuscular make-up. Nystagmus, usually consisting of lateral movements, is presented by over half the cases, perhaps in seventy per cent. True nystagmus, in which the eyes constantly oscillate, occurs about once out of five such cases, the other four presenting nystagmiform movements only on occasion, particularly when the eyes are forcibly deviated to the right or left. It should be sought by directing the patient's line of sight throughout the entire lateral and vertical range of ocular movement. In seventeen per cent. of cases Uthoff found *paralytic* conditions of the extrinsic eye-muscles. These, like the hemiplegic features, are usually transient. They may constitute a complete *lateral ophthalmoplegia*, or be confined to any of the motor oculi nerves, or to portions of the third cranial pair. They are, as a rule, *bilateral* and affect the *associated* muscles, showing their nuclear nature. The nystagmus is of a similar nature, and probably due to the disturbed muscular balance.

The pupils may show *minis*, inequality, and diminished reflexes to

<sup>1</sup> *Deut. Arch. f. Klin. Med.*,<sup>2</sup> Bd. lxxv, Nos. 1 and 2.



both light and accommodation, but the Robertson pupil is practically unknown, and miosis is usually associated with an exaggeration of the light reflex.

The optic nerve is very frequently affected and vision is often modified. The papilla may show *optic neuritis* or *atrophy*, but the atrophy is very seldom extreme, and the disc usually retains some color in a part of its expanse. Some papillary modification is found in over one-half of all cases, and some cases showing no changes in the nerve-head still present visual defects. Vision may be slowly or rapidly reduced and blindness may follow, but the amblyopia is ordinarily transitory, and a considerable degree of sight is frequently restored. Central scotomata, irregular or regular retraction of the field, *dyschromatopsia*, particularly for red and green, may be variously combined in the field, and in these respects the ocular disturbances are rarely symmetrical and may be entirely unilateral. They may be of insidious or rapid development and may be early or late manifestations of the disease. They bear a rough relation to the intensity of other symptoms and often increase correspondingly with them.

Visceral disturbances are insignificant in insular sclerosis. Rectal and vesical incontinence or retention, gastric crises, and other similar manifestations of organic disease of the cord are sometimes encountered, but are not severe in degree or constant in appearance.

Trophic disturbances are unusual. Loss of nails, glanular derciditis, and localized wasting of muscles, particularly of the interosseous muscles of the hand, have been met with.

**Bulbar Symptoms.**—Difficulty in swallowing and in mastication, trembling of the tongue, inability to protrude it, glycosuria, and polyuria are occasionally encountered, and, with the other bulbar symptoms already noted, point to the location of sclerotic patches in that region.

**Cerebral Features.**—Among the most characteristic symptoms of multiple sclerosis is the *speech defect*. This is very commonly present and consists of a *slow, monotonous, sonorous* pronunciation that slights no syllable and ends abruptly, *spontaneously*, explosively with the last. It sometimes causes elision of the final sounds or every syllable may be slurred. There is a distinct effort to articulate each sound with rests between. This staccato speech, in marked cases, is well marked, but in some instances it may lack some of its features or entirely default. Its mechanism is of difficult explanation, but is, at least in some cases, attributable to faulty action in the articulative muscles.

**Vertigo** is sometimes present and may disturb the patient at rest or in walking. In some cases the *astagnus* causes objects to dance before the eyes; in others, with ocular-muscle paresis, there is *diplopia*, both of which may cause vertigo. In rare instances the vertigo presents *Ménière's* type. The *apoplectic* and *galeiform* attacks that have been already incidentally mentioned, *spasmodic laughter* and *crying* and slight *mental encephaloid*, with *indifference* and *dejection*, indicate cerebral lesions. In rare cases an expansive, *egotistic delirium of grandeur* may appear closely resembling that of *parietic dementia*, and *complete dementia* may ensue. Hunt<sup>1</sup> insists that *parietic dementia* and multiple sclerosis are sometimes combined in the same subject.

<sup>1</sup>"*Ann. Jour. Med. Sci.*," Dec., 1903.

Hoffman,<sup>1</sup> in the 100 cases on which he based his study, found speech difficulties in 54, giddiness in 64, lurching in 40, optic atrophy in 50, nystagmus in 56, intention tremor in 71, parosmia in 66, and objectively disturbed sensation in 28. The sexes were about equally represented by 47 women, 53 men.

**Course and Forms.**—The onset may occur abruptly by an apoplecticiform attack, followed immediately by hemiplegic or paraplegic or monoplegic weakness, or these may suddenly appear without the convulsive attack. In other instances vertigo or visual disturbance suddenly opens the program. More frequently the onset is gradual and the course progressive. Difficulty in walking or in speaking or the trembling is first noticed and gradually increases, leading in the other prominent features of the malady. Sometimes the initial feature consists of attacks of pain of a stabbing, lancinating variety. The advance of the disease may be chronically progressive, or it may show exacerbations or remissions, and continue finally to advance. Sometimes the amelioration is permanent and recovery is possible. In progressive cases death may result from intercurrent maladies, from an apoplecticiform attack, from bullar accidents, or from the exhaustion of terminal desentia.

The duration of the disease, like its anatomical basis and its symptoms, is very variable. It may reach full development within a few months or a year, rarely almost at a bound. It may terminate in a year, last twenty, or recede completely at any time. Marburg, Bistocini, von Koch, and others have reported acute cases reaching a fatal termination in about three months.

In some instances only one or a few of the most notable symptoms of the disease ever develop. These cases constitute the *forma fronsa* of Charcot. Thus the trembling, or the speech defect, or the gait with or without nystagmus, or bullar symptoms and myotrophy may predominate, while other symptoms are only very slightly developed.

**Diagnosis.**—In a well-developed case the diagnosis may be made. The intention tremor, cerebellospasmodic gait, spastic weakness, nystagmus, and syllabic speech make a positive picture. The differentiation from *familial ataxia* may, however, present great difficulty when several members of the same family are affected with multiple sclerosis. Friedreich's disease is marked by flaccidity and abolished knee-jerks, the motor difficulty is purely ataxic and never spasmodic, and optic-nerve symptoms are practically always absent. In the heredo-cerebellar type of family ataxia there are increased reflexes and often optic atrophy, but a long family succession of cases rarely fails us, and the motor difficulty here again is devoid of spasticity and is purely ataxic. In neither type do we find the well-developed syllabic speech nor the intention tremor, though the speech is modified and ataxic and there are choreic movements which might at first mislead. Cases clinically mistaken for *myotrophic lateral sclerosis* have been recently reported by Prolet and Brauer, one for *transverse myelitis* by Simorling, and one for a *Benson-Steward paralysis* by Jeremias. The paraplegic cases may readily

<sup>1</sup> "Deut. Arch. f. Klin. Med.," 1910, Vol. 1, 2.

<sup>2</sup> "Wiener Klin. Wochenschr.," 1908, No. 34; "Kursch'sches Jour.," 1908, No. 4.



be mistaken for myelitis or spinal cord tumors, particularly as in some such instances segmental sensory disturbance is very definitely developed. Elsberg has done a laminectomy in such a case, and I have had a similar experience.

*Hysteria* is capable not only of mimicking every objective symptom of insular sclerosis, but sometimes is associated with it in the same patient. It should never be out of mind in making the diagnosis, and the stigmata of the neurosis should be faithfully sought in every instance. Cases showing hysterical signs and the symptoms of multiple sclerosis, according to Buzzard,<sup>1</sup> should be looked upon as probably organic if the tendon reflexes are exaggerated and, at the same time, the plantar reflex is abolished. This observer has noted optic atrophy in one-half of his cases of insular sclerosis, and found Babinski's toe-sign in all in which it has been sought. Absence or inequality of abdominal reflexes is also of decided significance.

In the partial cases, mainly marked by one of the prominent characteristics of multiple sclerosis, the diagnosis is decidedly difficult. In every such instance all the features of insular sclerosis should be sought, and some of them, if only partially developed, will be found to fix the diagnosis, or the presence of signs of other diseases will guide. When trembling predominates, we must exclude paralysis agitans, metallic intoxications, chorea, and hysteria. When the speech defect is most prominent, we must think of parietic dementia and the family ataxias. In cases showing a predominance of cerebellar ataxia intracranial tumors and the family ataxias must be excluded. When the gait is purely spastic, we must exclude transverse myelitis and the combined sclerosis of the cord.

**Prognosis.**—While the outlook in a case of multiple sclerosis is always grave, the tendency to remissions and the occasional complete recession of the disease make it distinctly more hopeful than in the destructive sclerotic lesions of tubes and cross-myelitis. It is impossible in a given case to forecast its probable course so long as it is progressing. Apoplectic seizures followed by paralytic features and aggravation of all the symptoms may occur at any moment; but when the disease has shown remission and improvement we are justified in hoping, with due reservation, for better things.

**Treatment.**—In every case the nature of the origin of the malady will guide therapeutics. If the disease is to be considered as mainly due to infectious, and to the continuous activity of microbial life, which from time to time leads to new plaques or increases old foci, bactericidal preparations are indicated—quinin in malarial; mercury, iron, arsenic, and salicyl preparations in other septic conditions. Generally speaking, an antiseptic regime is in order. This may only be valuable when the general resistive powers of the economy are increased by the best hygienic and general health measures. The future may furnish some organic antitoxin more powerful than our present medicaments.

<sup>1</sup>"Brit. Med. Jour.," May 6, 1899.





The *active specific lesions* of the general nervous system, like specific lesions elsewhere, originate in the connective tissue or blood-vessels and secondarily affect the parenchyma, disturbing function first and finally destroying the essential cells and fibers of the part. Rarely they arise in bony structures and thence invade the adjoining nervous apparatus or act upon it by pressure. Their peculiarity is in the gummy plastic exudate with its formative tendency and the presence of the parasite. It is essentially a neoplastic process, which may vary from an appearance of simple inflammation to the accumulation of sizeable tumors. In early stages there is often a marked tendency to regressions and recidives. Old lesions, if of considerable size, may encase within and circumscribe upon the surface by fibrous organization, and fibroid changes may alone remain to indicate the preëxisting gummy condition.

The *parasyphilitic lesions* are degenerative in character; such as the long posterior tract, degeneration of tubes, the optic atrophies, and the posterolateral so-called sclerosis. They seem to depend upon the continued activity of persisting spirochetes and are marked by glial increase in addition to the Wallerian degenerations.

The *active meningovascular lesions* are very amenable to treatment, while the parenchymatous degenerative lesions are so refractory as to be generally considered quite incurable. At best they may be brought to a standstill, the destroyed cells and fibers constituting a lasting defect, with persisting signs and symptoms. Antisyphilitic remedies readily reach and destroy the organism in membranes and vascular areas, but foci of infection within the parenchyma in the parasyphilitic cases are apparently impermissibly situated as far as known remedies are concerned, however administered.

Acquired syphilis affects the central nervous system by specific lesions in a very considerable proportion of all luetic cases. Hjeltnann, leaving out of the question both tabes and parietic dementia, stated that from 1½ to 2½ per cent. of all cases of syphilis develop cerebrospinal lesions, and that of those presenting the tertiary features of the infection, 12 per cent. show invasion of the cerebrospinal axis. Fournier put this latter percentage at 21.

That the extralaminar structures of the nervous system should be a favorite field of invasion for a disease notoriously prone to involve contiguous areas is not surprising. Orr and Rowe have suggested a lymphogenous route from the skin along the sheaths of the spinal nerves and the frequent root symptoms in early syphilis, and their relation to the usual location of primary and secondary lesions are highly suggestive. Changes of the spinal fluid giving positive serological tests with cellular increase have been determined in early syphilis in from 70 to 82 per cent. (Finger, Altman, Dreyfuss). It is a safe view therefore to consider the nervous system as probably directly affected in every case of syphilis and at an early period.

In the great majority of cases of nervous syphilis the process is widespread and somewhat haphazard in location. Very rarely do cases of spinal syphilis lack evidences of cerebral involvement at some period, but cerebral syphilis, on the contrary, is sometimes devoid of spinal complications. We may, for descriptive purposes, divide these cases

of acquired active syphilitic lesions into the cerebral and the spinal. Their common association should never be forgotten.

### CEREBRAL SYPHILIS.

**Syphilitic cerebral meningitis** is perhaps the most frequent form of cerebral syphilis, and usually, to some extent, complicates all other varieties of encephalic lues. The favorite location is the basilar area, especially the interpeduncular space, from which it frequently extends upward on to the convexity of the parietal, and particularly of the frontal, lobes. So commonly is the base affected that, speaking clinically and according to Charcot, it may never be considered except, even when the symptoms point to the convexity alone. The distribution recalls that of tuberculosis. A gelatinous transparent substance infiltrates the soft meninges about the circle of Willis and the optic chiasm. It is composed of small, round, embryonic cells, and is very vascular and viscid. Caseation and sclerotic degeneration mark the lesion later.



Fig. 486. Ocular injury in a case of brain syphilis, in which at one time there was a crossed hemiplegia, right face and left extremities. The right eye assumes, varying upward and inward its looking to the right.

The adjoining or enveloped nerves and vessels may be injured. Neuritic atrophy on the one hand and obliterating arteritis on the other are thus set up. Extension of the process to the nervo-sheaths and interstitial structure accounts for the very frequent symptoms on the part of the cranial nerves in cerebral syphilis.

In the field of syphilitic meningitis gummy masses, or *gummatæ*, are usually encountered. They vary in size from millary bodies to tumors as large as fillets, which may be solitary, few, or many in number. Their favorite location is at the base or on the convexity, especially on the frontal lobes, but no portion of the brain is exempt. Sometimes they form on the surface or in the thickness of the dura mater, or deeply within the brain, on some septal infolding or penetrating blood-vessel. They

comport themselves as do brain-tumors generally, and give rise to similar localizing signs and symptoms. Sclerous or fibrous degeneration marks the later stages of all these specific neoplastic lesions, and gives rise to what is sometimes called sclerous syphilitic meningitis.

**Syphilitic cerebritis** may exist as a direct extension from a specific meningitis or gumma, or may occur more or less independently. It may present itself as a diffuse, gummatous encephalitis, or as localized encephalic gummatæ which tend to circumscribe, or it may appear in circumscribed patches or plaques, especially in the pantine and peduncular surfaces, which may end in sclerous patches. Every syphilitic process in the brain entails a surrounding zone of cerebral softening or cerebritis.

**Syphilitic Cerebral Arteritis.**—Syphilitic endarteritis in the brain



is a comparatively common accident. It may result in the obliteration of the large basilar vessels secondarily to a gummatous meningitis, or it may develop as a primary focus of syphilitic activity. Its legitimate result is a localized ischemia, which, if complete, results in thrombotic brain-softening. Heubner insists upon its frequency in the basal ganglia. The middle cerebral arteries are frequently affected either in the numerous small branches of the Sylvian or by the complete obliteration of this motor-zone vessel.

It is also established that cerebral hemorrhages may follow a specific arteritis. This rarely occurs within the brain-substance, but more often takes place in the large and comparatively unsupported basilar vessels, resulting in a fulminant apoplectic stroke almost immediately fatal.

**Syphilitic Lesions of the Cranial Nerves.**—Owing to their exposed position at the base, where specific cerebral invasion is most common, the cranial nerves are very commonly implicated. The relative frequency with which they are affected diminishes from the olfactory and optic nerves backward toward the bulb. The order is as follows: Optic nerves, motor oculi, abducens, trifacial, facial, auditory, and only rarely the glossopharyngeal, vagus, and hypoglossus.

The motor oculi, or *Musci nerve*, is affected in about forty per cent. of the cases, and shows a particular vulnerability on the part of the filers supplying the levator of the upper lid. Hence the frequency and significance of ptosis. Inequalities of the pupil and pupillary stasis are of equal importance and frequency. A rigid pupil should always raise a suspicion of syphilis. It is in brain-syphilis that the Robertson pupil is to be met. Referring to the affection of the olfactory and optic nerves and tracts we find all forms of irregular and unequal contractions of the visual fields. Optic neuritis is very frequently present and atrophy may result. The trifacial may be involved in one or all its branches, causing disturbed sensibility sometimes, but more frequently neuralgic pains of corresponding distribution. Infrequently facial palsy is encountered, and in rare cases this is associated with deafness on the same side.

Undoubtedly, a careful examination of the optic and auditory nerves will find them affected in more than half of all cases during the secondary period of syphilis, as has been determined, among others, by Wile and Stokes.<sup>1</sup> The very general early implication of the cerebrospinal fluid is apparently ephemeral, but unfortunately permanent results are frequently entailed.

The amount of injury to a nerve varies within wide limits, but often presents the suggestive possibility of advance and recession, giving rise to temporary, *hyposecous paresthesia* and *transitory sensory parestheses*, such as amblyopia, lasting a day or two. After several such attacks the disability is likely to persist permanently and indicates a destructive change in the nerve, which commences its an interstitial infiltration and neuritis.

The general symptoms of cerebral syphilis are modified by the particular lesions that are present. These, as a rule, are *pathogenic*, but with special prominence of certain features. One case attracts most

<sup>1</sup> "Jour. Amer. Med. Assoc.," May, 1915, p. 1465.

attention by its eye symptoms, another by its neuralgic pains, another by its convulsive manifestations, another by its stupor or mania or paralysis.

There is always, or nearly always, an initial period—a *provisionary period*—that it is of the greatest importance to recognize. Then only does medication offer a fair chance of effecting a cure. Once passed, irreparable damage is done, and the best that can be hoped is to check further mischief and leave the brain and nerves scarred with cicatricial tissue. Brain-syphilis appears, ordinarily, during the secondary period, but may occur at any date after the first few months. Thus the first year shows the greatest number of cases, which diminish gradually, year by year, to the end of life. There is no period of exemption after infection. Cases of brain-syphilis are encountered twenty, thirty, and forty years after the initial sore, and probably many syphilized individuals die from other causes and thereby escape luetic brain accidents. If tertiary manifestations appear, the likelihood of the occurrence of brain-syphilis is increased. It is important to know that a blow or jar to the skull may precipitate and focalize syphilis in the brain, and may greatly aggravate it when present. The same is true of mental shock and fright. The phenomena of the provisional stage consist of *headaches, disturbances of sleep, mental changes, and physical decline*.

The headache in its typical form is atrocious in severity, often circumscribed, and deeply seated. It is frequently marked by local tenderness on percussion and shows diaily, usually nocturnal, exacerbations. The syphilitic headache may remit without apparent cause, or an exacerbation may be followed by a ptosis or an amblyopia, or it may terminate upon the appearance of a hemiplegia or other serious secondary brain-lesion. Usually the only sedative capable of controlling it is morphia, but it yields in a few days to either mercury or the iodids. While the headache is mainly vesperal or nocturnal, in some instances it is most severe in the morning and some trace of it usually lasts throughout the twenty-four hours. Commencing in a localized area, it may gradually invade the entire head, and frequently, if unchecked by proper treatment, lasts weeks and months.

The sleep is modified in two ways. There is *insomnia* or *aper*. The nocturnal headache in itself may destroy sleep at night, but in addition the patient often fails to sleep after the headache subsides. *Insomnia* often occurs early, before the headache has grown intense. Later, there is usually a tendency to *stuper*, and this also may appear early. The patient drops to sleep at any time during the day, at his meals, over his paper or desk. Aroused at night by the cephalalgia, he drops back into a heavy, stuporous sleep, and often requires vigorous rousing. In a somewhat way he may get up for any purpose and at once drop to sleep on returning to bed. The stupor may become continuous and reach a comatose depth.

*Mental changes* ordinarily accompany the cephalalgia. The patient's wits are dulled. He is apathetic, disinclined for study, work, or social enjoyment. The memory is frequently impaired. He looks and acts and feels depressed and indifferent. His ideas come slowly and he may show irritability or become greatly depressed.

The general health often fails. The appetite is commonly lost early,



strength diminishes, flesh is lost, and the skin and mucous membranes grow pale and anemic. Often at this time vertigo, tingling in an extremity, inequalities in the pupils, a drooping lid, or some twitching of a limb indicate the impending storm and show its direction.

Head and Fraenkeloides lay much emphasis upon *shiverings* and *nerve-root symptoms*. These are of frequent occurrence not only in early stages but in the late degenerative forms. The patient frequently complains of intense shivering on taking a bath or even on using a rough towel, especially on the dorsum of the body. A careful examination of sensation will usually detect nerve root, nerve trunk, or segmented areas of disturbance of a minor sort. These are most common in the lumbosacral field, next in the cervicofacial area, but may be very definitely intercostal or thoracico-spinal. In some cases paresthesias and neuralgias are pronounced and persistent.

**Special Symptoms.**—After the premonitory period or even from the first the disease shows one of three major tendencies, marked by the preponderance of *meningitis*, *arterial accidents*, or *lunar formation*, respectively. These may be and usually are variously combined.



Fig. 20.—Brain showing basal syphilitic meningitis, with granulated surface and small cysts in the deeper fissures (Dr. W. A. Jones).

The meningeal form is marked by extreme depression and torpor. The patient lies like one narcotized or stupefied with alcohol. At times he can be roused to answer in monosyllables, but promptly subsides into the lethargic state and may become unconscious and comatose for a few moments, for an hour, or for several days. When he talks it is in an uncertain and unreliable way. Frequently there is a little delirium. His headache alone seems to cause him trouble. Usually pupillary sluggishness and inequality are present, ptosis and squints are frequent, and diplopia sometimes admitted. There may be urinary incontinence

or retention. Fever is ordinarily, but not invariably, absent and the physical functions are well carried on. Death rarely results from this form of cerebral syphilis, but, on the other hand, complete recovery is equally rare. Some trace in the way of mental apathy is likely to persist. In exceptional instances there is wild, maniacal delirium and motor excitement, an elevated temperature, and quickened pulse.

**The Arterial Form.**—Cerebral syphilitic arteritis is usually accompanied by basilar specific meningitis and the premonitory headache. Cranial-nerve symptoms are also usually present. It gives rise to thrombus and cerebral softening, resulting in hemiplegia or monoplegia, or aphasia or other localized cortical disability. Usually this does not come on at once, but by slight premonitory strokes and numerous "warnings." Transitory plegias and aphasias are always suggestive of syphilis. When the motor cortex is involved, spasm or partial epilepsies are commonly induced. The rule applies here, as in gummatous, that epileptic seizures after the age of thirty are generally due to syphilis, and that paralytic seizures in adults, before the age of forty, in the absence of cardiac and renal lesions, are almost invariably syphilitic. Another indication of syphilis is a multiplicity of lesions. A double hemiplegia or left hemiplegia with aphasia or pseudobulbar palsy from double-sided lesions, or cerebral hemiplegia and paraplegia from brain and cord lesions, in the same case, speak for syphilis.

**Hæmorrhage**, as already indicated, is usually from a large basal vessel and is promptly fatal. It is commonly secondary to aneurysmal degeneration and is a rare cause of death in syphilitics. Occasionally thrombosis, beginning in the circle of Willis, eventually reaches and obstructs the basilar artery, and death follows coma.

The gummatous form of cerebral syphilis is usually marked by focal symptoms, and here all the rules of cerebral localization apply. As above indicated, the favorite location for gummatous is at the base and upon the frontoparietal convexity, precisely the regions in which focal symptoms arise. These consist of cortical states of irritation and destruction,—in other words, of limited epileptic attacks and cortical palsies, and of cranial-nerve lesions. Usually the focal signs and symptoms in gummatous cases are preceded by the premonitory phenomena. This might be expected, as the usual meningeal or cortical gumma is secondary in point of time to a more or less diffuse specific meningitis. In rare cases the convulsion is the first manifestation of cerebral invasion. Generalized fits and epileptiform convulsions are not rare in diffuse cortical syphilis, and may be associated with the Jacksonian attacks, petit mal, momentary aphasias, sudden "thickening of the tongue," and similar evidence of circumscribed lesions. Syphilis also seems capable, according to Fournier, of producing a parasyphilitic epilepsy to which attention will be recalled. (For further details regarding cerebral gummatous the reader is referred to the chapter on Cerebral Tumors, page 259.)

**Syphilitic Mental Diseases.**—The mental disturbances of brain syphilis are those of organic brain disease, but in subjects strongly predisposed to insanity the syphilitic cachexia may induce ordinary forms of insanity. Morbid mental states due to the encephalic ravages of lues



are most frequently of the depressed varieties grouped under the generic name of melancholia, with a decided tendency to dementia. Less frequently there is exaltation and delirium, which may be intense, and in both forms hallucinations are common. Signs of gross brain-lesions are rarely wanting. Cranial-nerve symptoms, localizing fits, monoplegic palsies, and speech defects point the way to diagnosis.

*General paresis*, paralytic dementia, or general paralysis of the insane is always postsyphilitic, and the spirochetes can be found in the cerebral lesions. Its full description, to which the reader is now referred, will be found in Dr. Peterson's portion of this work.

In some instances symptoms arise that closely ape those of parietic dementia, and give rise to the term *sypilitic pseudoparesis*. The differential diagnosis may, indeed, be impossible. The features suggesting active sypilitic disease, as contrasted with the degenerative changes of paresis, are a less steadily progressive course, less expansiveness, less amnesia, less speech difficulty, absence of the Robertson pupil, presence of cranial-nerve palsies and early monoplegias, and improvement under antisyphilitic treatment. Syphilophobia and hypochondriasis, arising from the knowledge or fear of being infected, have only an indirect relation to the disease.

**Diagnosis.**—The diagnosis of cerebral sypilis, when there is a plain history and marked evidences of previous infection, ordinarily presents no difficulties. In cases where such data are lacking, one of the rarest of diagnostic problems is furnished. Nocturnal headaches of the character described, epileptoid attacks occurring after the age of thirty, or apoplectic attacks appearing before the age of forty, should at once, singly or in combination, raise the suspicion of sypilis. A history of any sort of a genital sore strengthens the case; and if, in addition, the slightest secondary manifestation can be detected or recalled by intelligent cross-questioning, small doubt remains. It may be stated that the great majority of cases of sypilis of the nervous system present very slight secondaries, or these may have entirely escaped attention. Another indication of sypilis in brain disease is a multiplicity of lesions. Cord and brain symptoms in the same patient are significant, as are double-sided brain-lesions or lesions of both base and convexity. The almost constant presence of basilar gummy meningitis, with its consequent cranial-nerve symptoms, among which are ptosis, papillary abnormalities, and optic-nerve disturbances, often alone is sufficient to guide the diagnosis. The mode of development counts for something. In brain-sypilis the onset is rarely sudden, but is, rather, by repeated advances, with remissions. *Fragorosa pabula* and fleeting sensory losses are the rule in sypilis. The phenomena of the *prodomitory phase* can not be too strongly insisted upon. Sterility in male or female should be given a certain weight, and repeated miscarriages frequently irritate the husband.

Wassermann's positive reaction in the blood may be considered as strongly confirmatory of a clinical diagnosis when the other conditions which are capable of giving this reaction are or can be excluded. The spinal fluid in cerebrospinal sypilis, according to Nonne, presents the positive globulin reaction in every case: in from 90 to 100 per cent. of

takes and in from 95 to 100 per cent. of paresis. There is an increase of cells in the spinal fluid in at least 50 per cent. of early syphilis of the cerebrospinal area and in over 90 per cent. of the late or parasyphilitic conditions. The Wassermann reaction in the spinal fluid is positive in nearly every case if reliably made. These four reactions furnish positive evidence of the most substantial sort, but the clinical diagnosis is equally or more important. Finally, the therapeutic test stands for considerable. If, under adequate dosage, the disease yields within two weeks it sustains the diagnosis to some degree; but we must never forget that in other organic processes, even in sarcomatous tumours, the iodids may cause a notable remission in the symptoms.

**Prognosis.**—The outlook is far from favorable, though it is too commonly assumed to be so. It may be safely stated that less than one-half of the cases of brain-syphilis completely recover. Fournier, in 90 cases, reported 23 unrelieved, 13 improved, 14 died, and 30 recovered. Of those recovered, probably many presented recurrences later in life. In a general way we may expect to check the progress of active syphilitic processes excepting in rare cases of a malignant and unmanageable character, where the human organism can not tolerate a medication intensive enough to permanently subdue the disease. The golden opportunity is in the prodromal stage. Lesions that have caused destruction of nervous tissue, such as results from thrombotic softening, secondary degeneration, hemorrhagic disintegration, and sclerotic strangulation, leave indelible and irremediable effects. In view also of the known ability of syphilis to lie latent and dormant for years, and its clinical remissive tendency, the wise physician will always maintain great reservation in even the most promising cases. Marked and prolonged stupor and decided mental disturbance make the expectation of complete restoration very slight. A patient who has once developed syphilis of the cerebrospinal apparatus is never safe and must maintain medical supervision, and under the guidance of blood and spinal fluid tests should undergo courses of treatment at intervals as long as life lasts.

**Treatment.**—The treatment is the same as that in the spinal form, now to be considered, and will be detailed in that connection (page 501).

### SPINAL SYPHILIS.

Nearly if not every case of spinal syphilis sooner or later presents evidences of cerebral invasion, and the diagnosis of syphilis of the cord often depends upon this association. It is proposed in this section to mainly emphasize the cord-lesions.

Syphilis of the spinal cord and membranes is somewhat more precocious in its appearance than brain-syphilis, and most frequently appears from the third to the sixth month of the syphilitic history. It then diminishes in frequency during the rest of life. As within the skull, spinal syphilis is a disease of the meninges, nerve-roots, and blood-vessels. The cord-changes are invariably secondary and, in a sense, mechanical. It may involve any or all of the membranes as a specific meningitis, which sometimes is marked by gummatous thickening and tumor formation, but much less commonly than is the



case within the skull. The nerve-roots and pial vessels are frequently involved in the specific meningitis, which may also extend to the periphery of the cord. Another group of conditions arises from impairment of the blood-vessels. Endarteritis, endophlebitis, and thrombosis may cause disturbance of the circulation in any part of the cord. They may cause softening and changes that are clinically similar to a focal myelitis, or, by affecting the blood-supply of the posterior arterial field, set up the changes in the posterior-lateral portion of the cord that induce combined sclerosis. Ordinarily, spinal syphilis is multiform, and a varied picture is presented through the implication of various parts of the cordal apparatus. The matter is still further complicated by the variations in a single case, which may at different times present great modifications. Thus a case beginning as meningitis may become a meningo-myelitis, set up a combined sclerosis, and terminate by focal softening, consuming many years in its history. For descriptive purposes, and based upon clinical manifestations, we may divide cases of spinal syphilis into: (1) Meningitis and meningomyelitis, (2) acute myelitis, (3) ataxic paraplegias or combined sclerosis, and (4) tabes dorsalis, already described.

**Syphilitic Meningitis and Meningomyelitis.**—These furnish the most habitual expressions of spinal syphilis. In clinical features they differ but little save in the absence of fever from similar lesions arising from any other source. The principal symptoms are pains and palsies. The pains are onset features and are of extreme intensity, usually with a well-marked tendency to nocturnal exacerbation. They are usually located in the lower extremities, but with these there is generally associated a most intolerable *exhalgia* in the lower part of the back. They are *root pains* and may be marked by tenderness along the nerve-trunks, and even by herpes and glossy skin. Partial palsy of certain muscle groups, with disturbed sensation in the cutaneous area of identical innervation, is occasionally seen, and still further confirms the neuritic and root disturbance.

The *palses* are rarely complete. There is a feeling of weakness in the legs, sometimes distinctly greater on one side than on the other; a paraplegic distribution that may ascend to any level, but most frequently remains below the waist-line. In rare cases the upper members are most or alone affected. In some cases a polymeningitis cervicalis has been found, with its characteristic deformities, and in some such instances the spinal portion of the dural disease has been a downward extension from syphilitic thickening within the skull.

The cord is frequently affected, as is shown by bedsores, sphincter disturbances, Brown-Séquard paralysis, and a tendency to spasticity in the lower extremities, which are at first relaxed and caecobled.

The sensory disorders usually open the program and the paraplegia is ushered in by a feeling of heaviness and clumsiness in the legs. Usually there are a series of remissions and advances until the spasmodic paraplegia is finally established.

Occasionally, gummatous form in the meninges and give rise to tumor symptoms or the Brown-Séquard syndrome. Multiple miliary gummata are still rarer.

In the annular invasion of the cord the blood-supply is interfered with, both arteries and veins suffering from deformity and endothelial changes that may lead to ischemic softening and hemorrhage. Combined sclerosis are similarly induced.

**Syphilitic acute myelitis, or softening,** is not a rare condition. The best of recent observations indicate that the origin is in thrombotic occlusion of blood-vessels, syphilis acting as any other infection, or by setting up a specific arteritis or phlebitis, with or without accompanying meningitis. In the softened area there is commonly great syphilitic cellular activity, and secondary hemorrhage frequently causes a hematomyelic condition. The favorite location for softening is in the gray substance, and the thrombosis may extend to all the vessels of a considerable area. Very rarely does the lesion involve the entire cross-section. Secondary ascending and descending degenerations naturally follow.

The symptoms are those of an acute myelitis: sudden onset, paraplegia, corresponding sensory disturbances, sphincteric incontinence, and spasticity after a few weeks.

**Syphilitic Ataxic Paraplegia.**—This variety of spinal syphilis has been much written about since Erb, in 1892, called attention to its frequency and uniformity. He proposed for it the name of *syphilitic spinal paraplegia*. Subsequent communications by Mouchin, Kuh, Kozakowski, and others tended to give it an autonomy that Erb specifically deprecated in his earlier reports. Subsequently<sup>1</sup> he asserted for it the dignity of a distinct clinical type due to primary changes in the lateral tracts of the cord following syphilis in the same sense that posterior sclerosis is post-syphilitic. Oppenheim, Reissand, Marie, and others have denied its reality.

The condition is usually of insidious onset, developing in a few weeks, months, or years. The first symptoms are feelings of heaviness and paresthesia in the legs, very soon accompanied by difficulty in emptying the bladder through spasmodic action of sphincter and detrusor. Rigidity and spasticity in the legs soon develop, and the gait shows them in the dragging toes, abducted knees, and uncertain steps. Knee-jerks, reflex and ankle-contractions, and the toe-sign are commonly well marked and muscular strength is more or less reduced. Spasticity and a lack of muscular strength are especially insisted upon by Erb. Sensory disturbances are slight or entirely absent, and the disease is very slowly progressive or inclined to be stationary or to even improve under treatment for syphilis. It seldom reaches a stage of complete helplessness. Often the upper extremities are affected in a less degree, and papillary symptoms are not uncommon.

In this outline it is easy to see the symptoms of posterolateral sclerosis or ataxic paraplegia, or the condensed sclerosis (see p. 454). The condition may also arise secondarily from a meningomyelitis, and it is clearly due to vascular disturbance in the posterior arterial cord-field, with the ataxia, spasticity, and paraplegia that mark such lesions.

**Diagnosis.**—The diagnosis of spinal syphilis is frequently of the greatest difficulty. Unlike cerebral syphilis, it has not a characteristic

<sup>1</sup> *Br. Med. Jour.*, Oct. 11, 1902.



premonitory stage. The nocturnal neuralgia and the advance and retreat of the spinal symptoms have some significance, but a history or the evidence of syphilitic infection has more weight and the presence of encephalic disease has most of all. This last, as already frequently stated, rarely defaults. In the confined scleroses we must exclude tabes, which can usually be done by a tap on the knee and an examination for objective signs. Blood and spinal fluid tests have a very great value, particularly the finding of an increased cellular content in the cord fluid. To this commonly, almost invariably, are added Wassermann's positive in blood and spinal fluid and the positive globulin reactions. (See page 497.)

**Prognosis.**—The outlook in focal myelitis is the same as in that lesion from other causes than syphilis, and depends upon the amount of damage to the cross-section, the vertical location, and the secondary degenerations. Meningomyelitis can frequently be held in check and offers a good prognosis if the cord is only superficially injured. The prognosis of the confined scleroses is good as to life, but bad as to complete recovery.

**Syphilitic neuritis** is not a common accident. In rare instances the toxic condition may induce a multiple neuritis. Cestan<sup>2</sup> was only able to collect 14 cases from the literature, but in minor degree it is probably not infrequent. Usually single nerves, as the sciatic, intercostal, or some branch of the brachial plexus, are affected, and such lesions are often bilateral.

**Hereditary Cerebrospinal Syphilis.**—Hereditary syphilis is capable of provoking any and all of the injuries of the general nervous system that follow the acquired infection, and, like it, is often marked by a multiplicity of lesions in a given case. These may appear congenitally, in the early years of life, or be tardy and postponed until puberty, or even to the twentieth and thirtieth year. The presence or history of other syphilitic stigmata, such as Hutchinson's teeth, hydrocephalus, idiocy, cachexia, syphilitic skin diseases, choroiditis, and a history of parental syphilis, must often be relied upon for a diagnosis. This is confirmed by the characteristic blood and spinal fluid findings of cerebrospinal syphilis. The lesions of hereditary syphilis are less amenable to treatment and of worse prognosis than those of the acquired form.

**Treatment of Active Cerebrospinal Syphilis.**—The moment syphilis of the cerebrospinal apparatus is discovered, or even strongly suspected, intensive treatment should be instituted. The diagnosis of cord or brain syphilis implies the recognition of a critical condition.

The object of treatment is not only to cure the nerve lesions if possible, but to eradicate syphilis from the system. It may be admitted that this cannot often be fully accomplished. The postmortem findings of Warthin, however, indicate that the activity of syphilis may terminate, and that thereafter the specific organism remains imprisoned in the tissues. Moreover, destructive and degenerative processes cannot be restored, but we attempt to bring their progress to an end. As long as the lesions are of the meningovascular type, are inflammatory and exudative, we may expect to secure a substantial control and pro-

tical cure. When the organisms have invaded the parenchyma of the brain and cord and escaped from the fibrovascular structures, our present remedies seem incapable of reaching them in spirillum quantities.

The administration of the newer arsenical preparations by intravenous and intramuscular practice usually suffices to control syphilis in the nervous system, but it is often necessary to add courses of mercury and iodid. There are many who believe that the intraspinal or intracerebral administration of arsenic and mercury have additional advantages, particularly in the so-called parasyphilitic stage. I have not found this to be true in any degree commensurate with the difficulties and dangers of the methods employed.

A preliminary spinal puncture and a full examination of the spinal fluid and of the blood-serum is an important step to the proper management of any case of syphilis of the nervous system. The desired end is to render the blood and spinal fluid continuously negative to the specific tests. In the exudative forms of syphilis this can usually be accomplished with six or eight weekly injections of the salvarsan group of arsenicals, though an excess of globulin not infrequently persists. If the fluid does not tend to promptly become normal the salvarsan may be followed by a course of mercurial injections or intramuscular injections.

In the early stages of the treatment of the exudative type of syphilis the iodids are unquestionably of great therapeutic value, rapidly clearing away syphilitic deposits and probably facilitating the deeper penetration of the metallic remedies. For the degenerative type of syphilis of the nervous system the iodids appear to have so little value that their unpleasant effects on the stomach and skin may well be avoided.

Cases of tabes and paresis are undoubtedly often improved, remissions secured, and in the case of tabes the progress of the disease sometimes definitely stopped.

Guided by the spinal fluid tests or more roughly by the blood-serum condition, alternating or conjoint courses of arsenic and mercury are to be employed for long periods of months. But in the majority of cases of tabes and paresis no amount of treatment will secure a lasting negative condition of the spinal fluid, though the clinical condition may show much improvement. Kaplan<sup>1</sup> is of the opinion that the cases of tabes which remain Wassermann fast are likely to develop paresis, and the same thing is true of those cases in which the goldsol globulin reaction retains the so-called parietic curve. At intervals of six months and later of a year, and always under the guidance of the laboratory findings, courses of treatment should be administered to the end of life. Not otherwise can the subject of syphilis of the nervous system be fully guarded against the renewed activity of deeply colonized organisms.

In fighting syphilis we should never forget the patient. At times all specifics must be withdrawn and the general system built up. This is sometimes necessary where the limit of toleration appears to be reached and the symptoms of specific activity are still progressing. Supportive measures are indicated at all times. Ferruginous tonics, a liberal diet, massage, salt baths, and frictions are all valuable. The use of large quantities of drinking-water, hot or cold, and the employment

<sup>1</sup>"*Jour. Amer. Med. Assoc.*," Dec., 1903, p. 2234.



of hot baths, or Turkish baths, if they can be borne, are of assistance and often enable the use of larger doses of specifics than could otherwise be exhibited. Here arise the benefits of thermal springs and similar resorts, at which, in addition, the patient is often freed from domestic and business worries.

The results of cerebrospinal syphilis in the way of plegias and nerve-atrophies require the same management as when arising from other causes.

#### THE SO-CALLED PARASYPHILITIC DISEASES.

Aside from the ordinary lentic lesions of the brain and spinal cord, there is the long list of so-called parasyphilitic diseases. These are not marked by round-cell invasion and gummy process, nor are they so directly amenable to specific medication. They may develop hereditarily or, as is more commonly the case, follow acquired syphilis. A brief consideration is all that is required here, as they are individually dealt with elsewhere.

**Acquired Parasyphilitic Diseases.**—Chief among these are *tobes* and *paretic dementia*, which are always postsyphilitic accidents.

*Optic atrophy* is commonly due to syphilis, and like atrophy of the *eighth nerve* and the acoustic apparatus may constitute the principal manifestation of the degenerative type of syphilis of the nervous system for many years. A painstaking examination will usually detect other evidence of nervous syphilis. The same isolated degeneration may fall upon the anterior root apparatus and result in corresponding muscular atrophies.

In the *cachexia of lues* the general physical depravity furnishes a favorable soil for the development of *neuritis*, *epilepsies*, and *neurasthenia*, which do not differ from the same nervous arising independently of syphilis, but they are benefited or cured by the removal of the specific cachexia. They are usually attended by considerable mental depression, the mental equivalent of the muscular asthenia.

According to Fournier,<sup>1</sup> in addition to the epileptoid manifestations of cortical invasion, there is an *epilepsy* peculiar to syphilitics. Its characteristics he outlines as follows: (1) It is unattended by other evidence of cerebral disease; (2) it continues unchanged; (3) it is of long duration, even lasting the lifetime; (4) it is not amenable to antisymphilitic remedies; (5) the bromids have little control over it. Its onset is abrupt and usually in the form of a full epileptic seizure, without prodromata or inciting causes, and it often continues in the form of petit mal, grand mal, or a varying association of both. The attacks are commonly frequent during the first two or three years, after which they appear at long intervals only. Confirmation of this doctrine is lacking as yet, but cases corresponding to the outline are not extremely rare.

**Hereditary Parasyphilitic Diseases.**—In this category can certainly be placed the rare cases of *juvenile tobos* and *paretic dementia*. In addition all variations of *deficient vitality* and *defective growth* may be due to parental syphilis. Notably traceable to that source are *infantile paralysis*, *mental defects*, *idioty*, *hydrocephalus*, *optic atrophy*, and various *cerebral and spinal atrophic states*.

<sup>1</sup> "Les Affections Parasyphilitiques," Paris, 1894.





## PART VII.

### THE NEUROSES.

---

A LARGE number of diseases are clearly related to the nervous system and manifested on its part by disturbance of functional control. They are denominated *neuroses* or *functional nervous diseases*. It is better to avoid the term "functional," as it tends to distract attention from the anatomical basis of these diseases. There is little doubt that sooner or later they will be histologically classified. The present arrangement is for utility only. The following table serves to group the neuroses for descriptive purposes:

#### NEUROSES.

	Arteriosclerosis, Adipose diabetes, Adiposity with genital dys- trophy, Precocious sexual maturity, Myxodema, Exophthalmic goiter, Sclerodema, Tetany, Raynaud's disease, Acroparesthesia, Intermittent limping, Angio-neurotic edema, Hyperostosis cranii, Pulmonary osteo-arthropathy, Localized hypertrophies.
I. GLANDULAR NEUROSES.....	
II. VASCULAR NEUROSES.....	
III. HYPERTROPHIC NEUROSES.....	
IV. INJECTION NEUROSES.—Mainly marked by uricæ disturbance.....	Tetanus, Hydrophobia, Cholera, Huntington's disease, Myoclonus, Dührsen's disease, Parkinson's disease, Thomsen's disease, Writers' cramp, Occupation spasms, etc.
V. MOTOR NEUROSES.....	Neurasthenia, Psychasthenia, Hysteria, Epilepsy, Migraine, Tris
VI. FATIGUE NEUROSES.....	
VII. PSYCHONEUROSES.....	
VIII. NEUROSES FOLLOWING TRAUMATISM.....	

## CHAPTER I. GLANDULAR NEUROSES.

In the group of neuroses marked by disturbances of nutrition some abnormality of the vasomotor control is commonly present. This may furnish nearly the entire symptomatology, as in angioneurotic edema, Raynaud's disease, and exophthalmic goiter. In other instances the vascular element is apparently small, and the processes of nutrition are principally disordered. The relation of nutrition to vascularity, however, is always intimate. We can not as yet positively say through what individual strand of fibers trophic control is maintained, but it must be in close touch functionally with the vasomotor nervous apparatus. Certain groups of trophoneuroses are directly related to the chain of ductless glands: one group principally to the pituitary body, another to the thyroid and parathyroids.

**Trophoneuroses Related to the Hypophysis Cerebri.**—The function of the pituitary gland has been practically unknown until very recent years. Anatomically, it consists of—(1) a posterior lobe of nervous tissue; (2) a middle portion or septum of epithelial character, outgrowing from the buccal sac; and (3) of an anterior epithelial lobe of similar origin and constituting a remnant of the primitive or embryonic mouth parts. This anterior glandular portion formerly discharged its secretion into the mouth, and a rudimentary duct is still traceable in some subjects.

The posterior portion, the *pars nervosa*, furnishes a principle similar to the product of adrenalin that raises arterial tension. This secretion reaches the nervous apparatus through the infundibulum and third ventricle. It also seems to control fat accumulation. The anterior lobe, the *pars anterior*, has an intimate association with bony growth and with sexual activities. The *pars intermedia* appears to be related to the excretion of urine and possibly the control of sugar tolerance. The pituitary in its varied functions is also related to the activities of all the other ductless glands of the body, the thyroid, parathyroids, thymus, adrenals, islands of Langerhans, ovaries, and testicles.

Overactivity of the hypophysis, or hyperpituitarism, apparently causes the remarkable changes of acromegaly; underactivity, hypopituitarism, causes failure of development and asexual characteristics, sometimes with increased deposition of fat. These states are modified and clinical variants are induced by the period of life and the state of growth present when the morbid glandular condition becomes operative, and further differences are due to the degree of functional glandular perversion and to the varying degrees in which the diverse pituitary functions are affected. A series of cases with many overlapping features may, therefore, be encountered from one end to the other of extremes of hyper- and hypopituitarism. Life is probably not possible with complete loss of the hypophysis. In dogs apituitarism caused by the removal of the anterior lobe causes a fatal cachexia (Cushing, Paulsen).

The lesion causing pituitary disease is commonly a neoplasm beginning in the gland itself or in its vicinity. Thereby many "neighboring"



local symptoms are added, such as disturbance of the visual fields by the implication of the chiasm or optic tracts, especially hemianopia; headaches, and stuporous conditions from endocranial pressure, and distention of the sella turcica. In other instances new growths are not found, but glandular changes are present, showing particularly in cellular modifications.

As pituitary diseases we may tentatively group acromegalia, Froehlich's dystrophia adiposogenitalis, infantilism, and Dercum's disease.

#### ACROMEGALIA.

In 1886 P. Marie<sup>1</sup> called attention to two cases presenting acquired symmetrical enlargement of the hands, feet, and face, and proposed the



Fig. 304. Stature, muscularity, and symmetry in a patient twenty-one years old, wearing (under white shirt) Froehlich's (P. Marie).

name *acromegalia*, which has now become current. His description of these cases was so full that little has since been added to the clinical side of it. Similar cases were at once recognized all over the world. In 1892 Collins<sup>2</sup> was able to collect about ninety cases from literature. Since then the material has rapidly increased, and many autopsies have been put on record.

<sup>1</sup> *Berlin. Med.*, 1886.

<sup>2</sup> *Jour. Med. and Nerv. Dis.*, N. Y., 1892.

**Etiology.**—The causation of the disease is extremely obscure. Alleged or supposed inciting causes are almost as numerous as recorded cases, and embrace well-nigh every ordinary and exceptional experience of human life; consequently, no importance at present attaches to any of them. Both sexes are about equally affected. The disease makes its appearance customarily between the ages of eighteen and thirty. Exceptional cases have developed in advanced life and others in childhood. Several have appeared so early that they have been considered congenital. Occasionally brothers (Fraenkel) or parent and child have been similarly affected.<sup>1</sup> It has been found in association with tales, syringomyelia, nuchitis, gigantism, exophthalmic goiter, goiter, and various psychoses.

Klets, impressed by his first case in which the thyroid gland persisted, and Erb by the area of sternal dullness in other cases, were disposed to consider the changes in the bones and soft parts as due to a thymic angiomatosis. Later cases failed to present the enlarged thyroid, and the sternal dullness is probably sometimes due to thickening of the bone itself.

The usual adenomatous enlargement of the pituitary body and the frequent defective or goitrous condition of the thyroid have led to the belief that acromegalia is a trophoneurosis dependent upon modifications of the functions of these glands, especially of the pituitary. Rogowitzki, for example, considers that these glands destroy certain substances which have a toxic influence on the central nervous system. Others think that they secrete certain substances needful to the proper action of the trophic apparatus. All that can now be said is that the *prothypophysis cerebri* is nearly if not always hypertrophied, but whether as a cause or result is not determined. It is true, however, that tumors and other destructive conditions of the pituitary have been found without acromegalia.<sup>2</sup> That there is a definite relation between the pituitary gland and general growth and sexual development is quite evident from cases of pituitary disease associated with infantilism and defective genitalia. Such cases are reported by Hildevering, Fuchs, Haushalter, Lucien, and Froehlich.<sup>3</sup> Caselli contends that the pituitary gland is essential to life in dogs and cats, while Friedman and Marx<sup>4</sup> deny it. Israel<sup>5</sup> reports a well-marked case of acromegaly under observation for some years in which the pituitary gland was normal. Lewis<sup>6</sup> called attention to marked hyperplasia of the chromophile cells of the glandular portion of the hypophysis, which otherwise appeared normal, seeming to confirm the theory that acromegaly is caused by the excessive function of the glandular elements of this organ. The masterly studies, animal experiments, and operations of Cushing<sup>7</sup> make it evident that excessive activity of the pituitary is associated with abnormally increased growth, under- or hypopituitary activity, with retarded growth, and both with

<sup>1</sup> Fraenkel and Giglioli, "Nouvelle Iconographie de la Salpêtrière," Oct., 1908.

<sup>2</sup> J. de laet, "Virchow's Archiv," Bd. XLIII. <sup>3</sup> "Rev. Neurolog.," Jan. 15, 1909.

<sup>4</sup> "Berlin. med. Woch.," 1900.

<sup>5</sup> "Virchow's Archiv," vol. XLIII, 344.

<sup>6</sup> "Johns Hopkins Hosp. Bull.," Mar., 1908.

<sup>7</sup> "Ibid.," May, 1910.



genital inactivity. It is also evident that there are functional links between the pituitary, thymus, thyroid, and genital glands, and perhaps with the pineal gland.

genital inactivity. It is also evident that there are functional links between the pituitary, thymus, thyroid, and genital glands.

**Morbid Anatomy.**—The bones of the face, cranium, extremities, and to a lesser degree those of the trunk, show hypertrophy. The frontal and occipital bones are commonly thickened and their markings exaggerated. The frontal and maxillary sinuses are enlarged and the

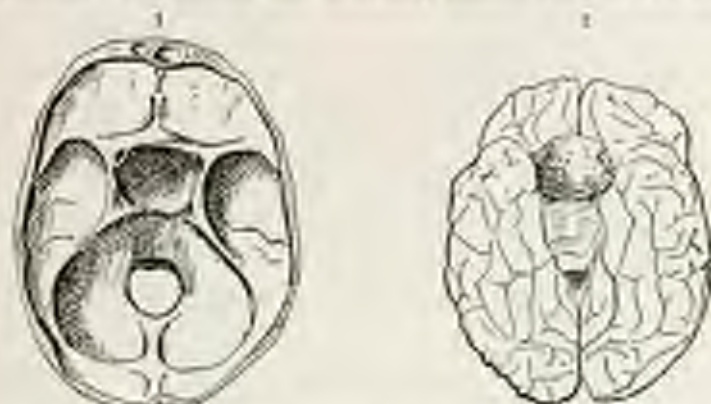


Fig. 244a, b, Base of cranium showing enlargement of the sella and 2, OPTIC CHIASM (Fig. 244a, b, acromegaly, "Necrot").

pituitary fossa is greatly increased in size. The inferior maxilla, the malar, the zygomatic arches, and the superciliary ridges of the frontal are particularly enlarged. There is commonly elongation of the spinous processes in the cervicodorsal region, and the bodies of the vertebrae are sometimes increased in the anteroposterior diameter. The ribs, clavicles, and sternum are commonly, and the shoulder-blades occasionally, enlarged. In the limbs the hypertrophy is most marked toward the distal extremities. The long bones of the leg and forearm are most enlarged at their lower ends. The metacarpals, metatarsals, and phalanges are particularly involved. Histologically, the hypertrophy is a true one, bone being deposited under the thickened periosteum and the central canal enlarged by the action of osteoblasts.

The pituitary gland is enlarged and hypertrophic, filling up the distended and enlarged sella turcica and commonly compressing the optic chiasm and adjoining structures. It practically constitutes an endocranial tumor. Strümpell<sup>1</sup> is inclined to think it an essential finding. In many cases it has been found to have undergone changes, probably secondary in point of time and nature. Tamburini<sup>2</sup> believes the first stage is one of enlargement and increased function, followed by adenomatous, sarcomatous, or cystic degeneration. A number of cases showing such degenerative changes are cited by Strümpell<sup>1</sup> and reported by others. In all cases the pituitary is diseased.

<sup>1</sup> *Deutsche Zeit. f. Nervenzellh.*, 1897.

<sup>2</sup> *Centralblatt f. Nervenzellh.*, Dec., 1894.

<sup>3</sup> *Loc. cit.*

In the extremities and other portions affected by the hypertrophic enlargement the skin in its epithelial, dermal, glandular, and muscular parts is hypertrophically exaggerated. The connective tissue particularly is increased. The subcutaneous nerve-filaments are sometimes degenerated as a result of the fibrous hyperplasia, but this does not extend



Fig. 32.—Radiograph of hands: A, (not of acromegaly); B, normal hand.

into the nerve-trunks. The skin is frequently overactive and the perspiration may be excessive and disagreeably fetid. In color the skin is usually ruddy, especially toward the extremities, and frequently marked with warts and pendulins, soft fibromata. The cutaneous sensibility is practically normal, but these patients are usually very sensitive to cold. The mucous membranes of the nasopharynx, larynx, and tongue are similarly affected. The kidneys often show a moderate chronic parenchymatous nephritis, with interstitial fibrosis. The spleen and lymphatic glands may be sclerosed; the thyroid is usually atrophic or goitrous; the thymus may persist and even be enlarged. It is to be noticed that, while parenchymatous glandular structures generally are atrophic, the pituitary is hypertrophic.

**Symptoms.**—Among the most common symptoms of acromegaly we may name hypertrophy of the face, hands, and feet, visual defects, dorsal kyphotic deformities, persistent headache, sexual failure in men, and amenorrhea in women.

The face is notably deformed. The greatly enlarged, overhanging brows are marked by sloppy, coarse eyebrows. Thickened eyelids; sometimes *exophthalmos*; a heavy, large, flabby nose; great *cherry-lips*; an enormous mouth, correspondingly thickened lips, and a much enlarged and prognathic lower jaw are some of the facial characteristics. The heavy features, puffy face, and accentuated nasolabial folds give an expression of *grief and suffering*. When the mouth is opened, it is seen that the



hypertrophic enlargement of the jaws, especially of the mandible, causes the teeth to stand separately and at considerable intervals. The mucous membrane is also thickened and the tongue is notably enlarged, in some instances reaching an enormous size. The soft palate, pharynx, and



FIG. 212 and 213.—Face in acromegalic two cases. Note enlarged superciliary ridges, thickened lips, massive jaw, and protruded glabella.

larynx equally share in the hyperplasia, and the tonsils and follicular structures are also increased in size. The ears and occiput are sometimes enlarged, sometimes not. The *nose* is thickened, the *hair* coarse, thick, and harsh. The *complexion* is usually yellow and the parts are elastically  *doughy* to the touch, showing no pits on pressure.

The *hands* are greatly enlarged in all their dimensions, but particularly in width. The *proportion* of fingers to hand and hand to wrist is retained; they are, therefore, enlarged symmetrically. The *fingers* are thick and sausage-shaped, the hand thick and beefy, the thumb and hypodactyl eminences greatly but proportionately enlarged. The whole hand is described as "spade-like or battleship-shaped." This appearance is sometimes intensified by a comparative thinness of the forearms. The joints are never limited in their range of motion, and the parts are surprisingly supple and flexible. The *polar furrows* are usually much deepened, the skin thick and resilient, the hairs coarse, the nails broadened, thick, and strongly striated longitudinally.

The *feet* show similar changes, and usually there is a heavy welt of fibro-elastic tissue around the heel and along the outer border of the foot.

The *thorax* usually presents an *anteroposterior spinal curvature* in the cervicodorsal region, which may be compensated by lumbar lordosis and attended by scoliotic twists. The *clavicles* are almost invariably increased in thickness and sometimes in length, and the *sternum* may also share in the hypertrophy. The *ribs* and *costal arches* are often enlarged. These bony changes give the chest an unusual breadth and antero-posterior depth. The *respiration* is ordinarily abdominal in character,

and the abdomen is often protuberant or even pendulous. The *pelvic girdle* may be enlarged.

The external *genitalia* in women are ordinarily hypertrophied, the uterus atrophic. In men there may be hypertrophy or atrophy of the external genitalia. *Ascensio* and *sterility* are the rule with women, sexual inappetence and *impotence* with men.

*Cephalalgia* of an intense, persistent, deep-seated character is present in the great majority of cases.



Fig. 214.—1 and 2, Hand, *in acromegalia*; 3, foot *in acromegalia*.

*Speech* is frequently thickened from the clumsiness and voluminous size of the tongue, and the voice is ordinarily very deep, strong, and rough, owing to the enlargement of the *larynx*, which to palpation may appear of increased dimensions and unusual prominence.

The organs of *special sense* may all be affected. *Sight*, *hearing*, or *tasting* may be reduced or abolished, but this is uncommon. *Vision*, however, is usually more or less affected eventually. This may arise from optic neuritis or from atrophy, and in either case points to encephalic tumor. Anislopia, blindness, intra-ocular pains, exophthalmos, contraction of the visual field, unilateral, homonymous, and bitemporal hemianopsia, and niasis have been noted. The bilateral loss of the temporal fields or their manifest reduction indicates injury to the chiasm by the pituitary tumor.

The *muscular system* may be normal or some atrophy may be found, but general muscular weakness is the rule. The *reflexes* are normal or diminished. The *diastolic responses* are frequently quantitatively reduced. Cardiac hypertrophy, arteriosclerosis, varicose veins, and slight lymphatic atrophy may be encountered. High arterial tension is the rule until terminal stages are reached, when a flabby heart and attacks of syncope are frequently encountered. The *thyroid* may be absent, normal, or hypertrophic. Polyuria, glycosuria, polydipsia, excessive appetite for food, and dyspepsia are common.

General *physical feelings* are usual, and *acral dysesthesia*, irritability, and *stupor* are common.



**Course and Forms.**—The disease is of insidious onset and slow progression, presenting a duration of twenty to thirty years and terminating by a cachectic state marked by great muscular weakness, during which death often occurs suddenly through cardiac failure. Intercurrent affections find acromegaly very vulnerable.

We may distinguish *two varieties* of acromegaly, depending mainly upon the age at which the disease commences and the condition of epiphyseal ossification with the diaphyses: (1) Before complete ossification takes place the enlargement is not only in lateral dimensions, but also in length, and the limbs become dis-proportionate to the body, producing gigantism. Autopsical findings by Hasselmann, Dani, Brewster, Taruffi, and skiagraphical investigations by Marinaccio conclusively show this relation. (2) After the diaphyses and the epiphyses are united by bone, only circumferential increase is produced, and the stature, from scoliosis, may actually be reduced. The commoner anatomic type originally described is then developed.



Fig. 155.—Acromegaly, with gigantism; anterior view, and pronounced distention of the abdomen (Marinaccio).

**Diagnosis.**—The diagnosis can rarely present difficulty if the disease has attained any considerable degree of development. *Marfanoid* statur is associated deformities, and the thickening of the soft parts is waxy and boggy. *Polyosteoarthritis* characteristically presents joint changes and a chronic polymeric process. The distal enlargement of the fingers is largely confined to the terminal phalanges, over which the hypertrophic nail-like parrot-beaks. In *hyperostosis cranii* the hands, feet, and mandible are not affected. *Paget's disease*, or *osteitis deformans*, spares the face, but affects the skull, and causes bowing of the long bones, especially the femora and tibiae. The x-ray commonly shows excavation of the sella in pituitary disease.

**Prognosis.**—The progress, as above indicated, is toward physical helplessness and mental depression, which in more than one instance have led to suicide. According to Thompson,<sup>1</sup> the disease may run an acute, intermittent, or chronic course, lasting from two to thirty years. Sudden death from cardiac failure is rather frequent.

**Treatment** is directed to relieving the cephalalgia and correcting the symptomatic disturbances as far as possible. For the first, phenacetin and similar synthetical analgesics seem to be most efficacious. Treatment by thyroid or thymus, singly or combined, seems to be of little avail, except in some instances to reduce weight, and then often at the expense of the general health and strength. The surgical removal of a part or of all of the anterior lobe of the pituitary gland, or of the pituitary tumor, has been made possible by the brilliant achievements of Horsley, Schief-fer, Esselberg, Kammer, Habot, and Cushing. Operation is indicated

in those cases which present marked symptoms of advancing brain tumor, particularly optic neuritis, or beginning optic atrophy and threatened blindness. It may also be undertaken to control the general acromegalic state, as already several operated cases are recorded in which there has been an actual recession of the bony and soft tissue enlargements. After hypophysectomy, the patient's condition must be watched and hypophysis feeding employed if the cachexia of a hypopituitarism appears.

#### ADIPOSY AND GENITAL DYSTROPHY.

The relations of the genitals to the pituitary are abundantly established by clinical observations and animal experiments. Anomorphia in female and impotence in the male subjects of acromegaly is a common observation. Some of the youthful giant cases never develop sexually. Castrated bulls and cocks show pituitary enlargement, and, on the other



Fig. 216—X-ray picture of normal sella turcica, marked.

hand, dogs subjected to partial excision of the hypophysis cerebri become fat and asexual with atrophic genitals (Cushing).

Juvenile cases presenting general adiposity and undeveloped genitals have been of occasional mention in literature, but Froehlich<sup>1</sup> first called attention to their relation to pituitary disease. Marburg<sup>2</sup> divides such cases into three classes: (1) Simple adiposity, (2) adiposity with genital atrophy, (3) simple genital atrophy. To these must be added (4) infantilism, or a simple lack of physical and sexual growth (Church<sup>3</sup>). Marburg further enunciated the formula that hyperfunction of the pituitary resulted in acromegaly; hypofunction in general adiposity and genital dystrophy, complete pituitary defect in a severe cachexia analogous to that after destruction or ablation of the thyroid. All these juvenile conditions are related to a lack or, in some instances, perhaps to only a perversion of pituitary control. Some of them present distinct symptoms of a pituitary tumor, such as hemianopsia, cranial nerve palsies, and the diffuse symptoms of brain tumor, especially headache, vomiting, and convulsions. The x-ray plate (Figs. 216 and 217) often

<sup>1</sup> Wien Klin. Wochenschr., 1908.

<sup>2</sup> Deutsch. Zeitschr. f. Nervenhkr., 1908.

<sup>3</sup> Jour. Amer. Med. Assoc., 1910.



gives a vivid picture of excavation of the sella turcica. A pituitary tumor is not necessarily present. This gland may be compressed by an adjacent growth or influenced by a variety of pathological conditions. Similar conditions arising after the attainment of full sexual growth cause, in varying degree, sexual inaptitude, genital atrophy, impotence, and menorrhoea, and may cause more or less obesity.



Fig. 37.—Lateral picture of sella turcica, marked  $\times$ , compressed by pituitary tumor. No sella markedly present.

The treatment must be carefully individualized. Brain tumor conditions must be treated on their own surgical indications. Decompressive operations are advisable in some instances. When the tumor is definitely limited to the pituitary, it offers an opportunity for a brilliant operation and an equally brilliant result. The cachexia resulting upon a lack of pituitary function must be combated by pituitary feeding, and this may be tried in the infantile or juvenile cases.

**Adiposis Dolorosa.**—In 1892 F. X. Dercum,<sup>1</sup> under the title *Adiposis Dolorosa*, described a condition which seems in the adult to be analogous to the adiposity and genital dystrophy of Froehlich which occurs in earlier years. This condition has come to be generally known as Dercum's disease.

In the **etiology**, neurogathic heredity or a personal neurogathic condition have not been noted with sufficient frequency to make them highly significant. Women are more often affected than men in the proportion of 6 to 1. The condition develops usually between the ages of thirty-five and fifty, though cases, both younger and older, are recorded. Women who have passed the menopause furnish the largest number. Alcoholism and syphilis have been present in a considerable number of instances, and their action in producing degeneration of the ductless glands seems to be established.

**Adiposis dolorosa** is of insidious development. The principal **symptoms** are a sensitive fatty deposit, pain, general asthenia, genital disturbances, and psychic disorders. The fatty deposit may be nodular, circumscribed, or diffuse. Some of the patients attain great weight, but

<sup>1</sup> "Ames Jour. Med. Sciences," 1892.

In many the body weight is not materially increased, especially in the nodular form. The fatty deposits are most commonly found over the trunk, shoulders, arms, and thighs, forearms and legs being less frequently affected, hands and face rarely. The fatty masses show a peculiar tendency to break readily. They present the peculiarity of being sensitive to manipulation and touch and frequently the seat of sponta-



Fig. 113.—Adipoma Abdominis of the Nodular Form (Harvey). The anterior view shows the peculiar aspect of fat and the small size of the hands. The posterior view shows the arrangement of fat in folds over the legs.

neous pains. The nerve trunks ordinarily are not sensitive, but may be so. Diminished sensation and paresthesias are frequent. The patients complain of numbness, coldness, crawling, tingling, etc. General weakness is commonly complained of. In both sexes sexual activities tend to subside in a degree out of proportion to the age of the patient. The mental changes are various, embracing depression, loss of memory, melancholia, delusional insanity, and dementia. Epileptic convulsions, blind-



ness, deafness, and various organic changes in the central nervous system have been incidentally noted. The tendon reflexes, commonly diminished or abolished, sometimes are increased. Vaso-motor disturbances are frequently encountered. Flushings, cyanosis, transitory edema, spontaneous hemorrhages from nose, stomach, and uterus, and absence of perspiration are not uncommon. Trophic changes, such as ulceration, the formation of blebs and bullae, arthritic and bony changes, have been noted. Headache, dyspnea, rapid heart, insomnia, and tremor are not infrequent, and the symptoms of fatty heart may be expected.

As to **pathology**, the reports of eight autopsies collected by Price,<sup>1</sup> whose description is mainly followed herein, indicates involvement of the ductless glands in all in which they were examined. The thyroid, while commonly involved, at least in one case was normal. In all of the cases in which the pituitary was microscopically examined, it was found to be diseased either by neoplastic formation or cellular change or round-cell infiltration. Dercum is now disposed to look upon the disorder as one due to hypergranularism.

The **diagnosis** is usually readily made if one bears in mind the association of painful conditions with deposits of fat, which are sensitive to pressure. The condition lacks the bony changes of acromegaly, and the painless, symmetrical, and uniform development of fat in ordinary obesity. The condition is not infrequently mistaken for rheumatism and frequently for multiple neuritis. The tendency of the disease is to permanency, and it does not directly cause death. Intermissions are rare, but remissions are common, and recovery has occurred only in one case (Dercum).

The **treatment** of adiposis dolorosa by the administration of thyroids seemed to result successfully in the one case reported cured by Dercum, and improvement has been reported in a number of other cases under the same treatment. The administration of pituitary extracts or pituitary feeding is indicated. When a pituitary tumor can be detected, surgical treatment should be considered. Alcoholism and syphilis must meet their appropriate management, and the condition of the heart, with its tendency to fatty degeneration, requires supervision. The pains and tenderness are generally beneficially affected by the salicylates.

**The Pineal Syndrome.**—The human pineal gland though long considered as a vestigial sense-organ of sight has more recently been found to have glandular constituents and to be related to the chain of endocrine glands.<sup>2</sup> It probably is active in early growth processes, but normally undergoes involution which begins at about the seventh year and is completed at twelve or fourteen. It seems to have special relation to bodily nutrition and growth, genital development, and the deposits of subcutaneous fat.<sup>3</sup>

It has been proved that pineal gland substance taken from young animals, such as bullocks and sheep, and fed to young chickens, guinea-pigs, and rabbits, causes a more rapid growth than is attained by con-

<sup>1</sup> *Amer. Jour. Med. Sci.*, May, 1909.

<sup>2</sup> F. Tilley, "New York Med. Jour.," May, 1919, p. 887.

<sup>3</sup> Datta and Barkley, "N. Y. Med. Record," May, 1911, p. 535.

tol animals, with a correspondingly earlier sexual development and reproductive capacity.<sup>2</sup>

Tumors or disease of this gland in prepuberal years sometimes brings about a remarkable early and abnormal growth of the whole body, but particularly of the genitals, pubic and body hair, and subcutaneous fat. The child of four or five attains the stature of one of ten or twelve, a deep voice, and a maturity of mind as shown by speech and thought. In addition, tumors of the gland give rise to the general symptoms of brain tumor and to neighborhood disturbances, especially hydrocephalus. Papan calls this pineal syndrome macrogenitosomia. It is not unlikely that other of the ductless glands and particularly the pituitary take part in this syndrome. It is suggested that disease of the pineal may occasion either glandular hyperactivity, or by disrupting the endocrine balance cause the trophic peculiarities of the syndrome.

Pineal gland preparations have been employed to stimulate both bodily and mental growth in backward children, but thus far without very definite results, though with some encouraging observations (Dana).

#### TROPHONEUROSES RELATED TO THE THYROID AND PARATHYROID GLANDS.

Among the ductless glands the function and diseases of the thyroid are of the greatest importance. Reduced functional activity of the thyroid is correlated with a group of dystrophic conditions and nervous and mental symptoms which finds a type in myxedema. Complete athyroidism, as, for instance, that produced by ablation of the thyroid, produces a fatal myxedematous cachexia. Lesser degrees of thyroidal inactivity give a large range of variations, embracing cretinism and infantilism when occurring early in life, and slight myxedematous disorders at all ages. Thyroidal overactivity finds its typical clinical manifestation in exophthalmic goiter, a condition again that has a wide variation of degrees of intensity.

Through the functional relation of the thyroid to the other ductless glands the various clinical manifestations of imperfect or disturbed thyroïdation are variously complicated by association with acromegaly, Addison's disease, dyspituitarism, and abnormal thymic states. Sexual abnormalities are commonly also present.

The various members of the chain of ductless glands apparently are able to substitute one another to a certain extent. They are also able mutually to stimulate each other, as has been proved by animal experiments and observations in disease by means of implantation and glandular feeding.

#### MYXEDEMA.

Under the generic term of myxedema it is proposed to bring together those clinical variations of nutritive disorder dependent upon partial or complete cessation of thyroid activity. In 1873 Gull described a *cretinoid state occurring in adult women*. Four years later Orl reported additional cases and proposed the word *myxœdème* (mucous swelling). Charcot, about this time, struck by the cutaneous thick-

<sup>2</sup> McCord, "Jour. Amer. Med. Assoc.," July, 1914, p. 232.



ing and the cachectic state, used the term *psychomyxedema cachectica*. In 1880 Bouverville and d'Ollier described a case of myxedematous idiocy and subsequently many more. In 1882 Reverdin, and shortly afterward Kocher, reported myxedema following complete extirpation of the thyroid, for which they severally proposed the terms of *acquired myxedema* and *cachectic strumipoint*. The analogies between acquired myxedema, myxedematous idiocy, and cretinism have been noted by many, and Brossaud, in his lectures of 1893-'94, brings certain cases of *defectilis*, or physical retardation, into the same category. We will commence by a description of acquired myxedema in adults. Due allowance being made for the age and growth of the individual, and the easiness and completeness with which the thyroid is affected, will



Fig. 255.—A case of myxedema before and after two months' treatment by thyroid (Dr. John Woodman).

practically enable this description to be applied to all the above-mentioned varieties.

**Acquired myxedema of adults** is of insidious onset, as a rule, and more common in women than in men. It usually appears between the ages of thirty and fifty. Occasionally it has abruptly followed an attack of acute rheumatism, some infectious fever, or a severe hemorrhage. It is marked by: (1) Tumefaction of the dermal and mucous structures; (2) by intellectual and physical enfeeblement, and (3) by atrophy of the thyroid body.

The *dermal changes* are the most striking. The skin is infiltrated with a mucoid substance and the fatty panniculus is frequently greatly thickened. The swelling offers an elastic resistance to the touch and

does not pit on pressure. The color is usually pale, yellowish, and waxy or cheesy. The face is enlarged, rounded, and apathetic. The heavy, thickened lids droop over the eyes, the nose and lips are thickened, the nose is furrowed, the ears enlarged, and the cheeks rounded and showing jelly-like trembling on slight jarring. The whole appearance is one of heliotele and stupidity. The skin of the trunk and extremities is likewise infiltrated. In the axillæ and subclavicular depressions lipomatous-like masses are often encountered. The hands and feet are enlarged with cushion-like swellings on their dorsal surfaces; the digits are thick and clumsy. The scrotum is often much thickened. The epidermal structures are greatly affected. The skin is dry, harsh, and branny. The hair becomes dry and scanty on all portions of the head and body. The nails are dry, fragile, striated, atrophic. Perspiration and sebaceous secretions are defective. The mucous membranes, wherever visible, are tumefied, pale, dry, and elastic. Mucous polypi in the nasopharynx are not uncommon. The tongue is thickened and, with the infiltrated condition of the buccal, pharyngeal, and laryngeal mucous membranes, explains the muffled voice and difficulty of swallowing.

The *intellectual state* in myxedema is uniformly marked by apathetic enfeeblement, and cerebral torpor shows itself in sluggish mentation, defective memory, slow speech, and lethargic movements. Myxedemic patients are usually indolent both mentally and physically, and are irritable and somnolent. Some cases show nocturnal insomnia and are troubled by disturbing dreams. The sluggishness of movement and the clumsiness of the hands and feet are due to the cerebral torpor and the local thickenings. These patients have no muscular energy and are promptly fatigued on the slightest continuous effort, but there is no palsy.

The thyroid in the great majority of cases can not be detected by palpation. There may be a history of its former presence or actual enlargement, and even a goitrous condition may persist. In some instances exophthalmic goiter has preceded myxedema and they have also been found associated, but Graves' disease does not follow myxedema.

Less prominent and constant conditions in myxedema are: cardiac weakness, irregular pulse, hemorrhages (especially metrorrhagia), a sub-normal temperature, loss of teeth, habitual constipation, and occasionally albuminuria in advanced cases. The patients complain of headaches, vertigo, throbbing in the ears, and particularly and almost constantly of a sensation of cold. Sensations objectively, motor conditions, and the reflexes are normal.

Usually insidious in onset, the disease runs a slow, tardy, progressive course. Remissions of longer or shorter duration may occur, as in summer, or by removal to a warm climate, and pregnancy sometimes has a similar retarding effect. The general tendency is toward encephalic helplessness and death by marasmus. In the very last stages the tumefaction may disappear. Pulmonary complications, especially tuberculosis, are common, and cardiac atrophy may strike the final note. Fortunately, treatment is now equal to the requirements of these otherwise hopeless cases.



**Operative myxedema** is usually the result of the total extirpation of the thyroid, which it follows in from three to six months. Removal of a part of the thyroid, the remainder being completely disabled,—by cystic disease, for instance,—results in myxedema. This is initiated by lassitude, physical enfeeblement, sensations of cold, heaviness in the limbs, sluggish and clumsy movements. Shortly the integument swells and becomes discolored, the hair falls, and cutaneous functions lag. The cerebral torpor follows and myxedema is fully established. Tetany may also develop, due, according to Murray,<sup>1</sup> to the loss of the parathyroid bodies, which in man are sometimes included in the lobes of the thyroid. The relation of the parathyroids to tetany and their control of the calcium metabolism is definitely established by McCallum and Voegtlin.<sup>2</sup> The course is usually progressive, but is more subject to remissions than in the spontaneous variety. Other cases improve, owing to the compensatory action of unremoved portions of the gland,



FIG. 256.—Myxedema, before and after thyroidectomy. 1, Five years old; 2, one year later (Parker).

or to the development of accessory thyroids, or through the vicarious activity of other glandular structures. The gravity of operative myxedema is great in proportion as the patient is young. Occurring in childhood, or at any period before adult life, it retards or completely checks growth, and produces a persistent infantilism or a myxedematous idiocy. It is, however, completely amenable to the thyroid treatment.

**Congenital myxedema, myxedematous idiocy, or sporadic cretinism** is usually first noticed at about one year of age, or upon weaning, and thereafter presents all the characteristics of adult myxedema, excepting that the mental faculties never develop and physical growth is retarded to the last degree. It is frequently congenital, and Horsley has found it in a dead-born fetus. It is encountered in the offspring of degenerate, alcoholic, or phthisical parents. At twenty years

<sup>1</sup> *Brit. Med. Jour.*, "Mar. 6, 1920." <sup>2</sup> *Johns Hopkins Hosp. Bull.*, "Mar., 1908."

of age these cretinoid idiots may have little more than two feet of stature. The relatively normal size of the head contrasts with the dwarfish body. The skull is full behind, contracted and narrow in front, often with persistent fontanel. The flabby, thickened features, snubbed nose, thick lips, drooping eyelids, mouth agap, rolling, hypertrophic tongue, and drooling saliva make up a peculiarly repulsive appearance. Add, *vis*, the short, often liponotous, neck; a protuberant abdomen, often showing inguinal and umbilical ruptures; a deviating spine, rudimentary genitals, and dwarfish, crooked limbs, and it is impossible to imagine a less human-looking object with human attributes. Sparse hair, eczema, and an infiltrated, inactive skin are commonly present. The thyroid is absent. Idiots mentally, they can ordinarily exercise a little attention and even show some affection. In some instances they assist themselves in eating and dressing. They never learn to speak, and never show signs of pubescence. Often even the first dentition is extremely defective. On the other hand, they lack the destructiveness, noisiness, fits, convulsions, onanism, balancing and motor disturbances so common in idiocy from cerebral lesion. These unfortunate creatures may attain thirty or forty years of age, and usually die of pulmonary circumstances.

The disease is also amenable in some degree to the thyroid treatment.

**Cretinism** is a term of ancient lineage and honored usage, but of somewhat uncertain definition. It has been applied to the goitrous and feeble-minded natives of localities where goiter is endemic. Certain valleys in France, Spain, Italy, and Switzerland, and some parts of Great Britain, Sweden, and of other countries widely scattered over the globe present endemic conditions that predispose to goitrous enlargements. Such an endemic has been noted in Minnesota and Ontario. A few definite facts are the results of observations extending over generations in some of these communities. The offspring of two goitrous parents, according to Kocher, is invariably a cretin, who may or may not be goitrous, but is myxedematous. A non-goitrous cretin invariably has goitrous antecedents and is indistinguishable from the myxedematous idiot or sporadic cretin, the condition also being congenital. In the goitrous cretins the thyroid disease may appear at any period of life, and acts then, exactly as does spontaneous myxedema or operative myxedema, to stunt growth and stop mental development. The goitrous cretin is usually also myxedematous and may present any degree of mental impairment, from mere simple-mindedness to abject brutishness. The distribution of endemic cretinism is identical with that of endemic goitrous disease, and Kocher believes this to be due to organic infections through the water-supplies. The water origin of goiter was strongly supported by Bircher, but a more recent survey of the question, and particularly in Switzerland by Dieterle,<sup>1</sup> discredits this idea. It is found that certain houses and families exhibit repetitions of the disease suggesting localized infection. Even hereditary relationship could not be demonstrated, the children of

<sup>1</sup>"Arch. of Hyg.," 1913.



goitrous parents being subject to the same circumstances of environment as their elders.

The only distinction between endemic cretins and other myxedemic patients is the goitrous enlargement. This may be only a difference of degree, because the cystic degeneration and interstitial hypertrophy at the bottom of the goitrous enlargement of the thyroid is destructive in character and effect. It is easily conceivable that myxedema and mental disturbances will be developed proportionately to the lack of functionally active thyroid. When the thyroid is entirely wanting, as in the non-goitrous congenital cretin, or completely destroyed in some goitrous cretins, the myxedema is correspondingly intense and the mental degeneration proportionately developed. It would seem, therefore, that the causes of endemic cretinism are those that produce endemic goitrous disease, in which the cretinoid state is secondary.



Fig. 221.—Myxedematous cretin eighteen years old (Parker).

#### Infantilism and Myxedematous Retardation.

Occupying a middle ground between myxedematous idiocy and acquired myxedema there are numerous cases showing slight cutaneous tumefaction, retarded mental development, and diminished growth. These patients retain their childishness both in mental attributes and physical conformation. Perhaps here belong some of the idiots classed as Mongolian by the English, and some of the *enfants gâtés* of the French. In at least one well-marked instance Brisson found the thyroid body scarcely perceptible. At the age of ten numerous and large cervical glands had been removed, with probable resulting damage to the thyroid. Thereafter the physical, genital, and mental growth of the lad had remained stationary. Schmidt<sup>1</sup> has treated three cases of this sort in which growth was retarded by the administration of thyroids, with immediate improvement. In such mild cases of cretinism the pituitary gland is frequently enlarged and there is often a mingling of thyroidal deficiency and dyspituitary activity with polyuria, excessive sugar tolerance, genital retardation, etc.

**Etiology.**—If we look upon myxedema as the manifestation of defective thyroidization, its causes are those of disease or absence of the

<sup>1</sup> "Thyroparacetic Wucher," *Nerv.*, 1896.

thyroid body. In some cases it is a *hereditary defect*; in others it is the result of a *thyroiditis* which may be dependent on infectious processes, as the infectious fevers, rheumatism, etc.; in others it is *cystic degeneration*; in others the result of *trauma* or surgical *extirpation*. Taking all cases of myxedema together, there is a preponderance of



Fig. 221.—Myxedematous children, youth  
distended (girls) and (distended).



Fig. 222.—Oreosoid girl of myxedema, 4  
feet tall, 92 pounds weight; distended and palpable;  
with teeth prominent; with various epiphyseal  
epiphyses compactly; usually backward; some-  
times indurated.

*frons* which reaches large proportions in the spontaneous adult varieties. It is probable that the close relation of the thyroid and uterine functions is at the bottom of this fact. It is only necessary to mention the increased size of the thyroid in pregnancy, its frequent enlargement in pubescent girls, its usual congestion in some women during menstruation, and its final retraction at the menopause. *Erysipelas* of the neck and head and *sycosis* of the thyroid gland have induced myxedema.



Regarding the manner in which defective thyroxination affects the trophic apparatus and induces the mucoid deposits, two general points of view depending upon opposite physiological hypotheses are maintained. As yet all is theory. Many, with Schüff, believe that the normal thyroid elaborates some substance indispensable to the proper action of the nervous system. This substance, however, has never been isolated nor its characteristics determined. Others, with Horsley, conceive that the thyroid transforms the mucinoid elements of the blood



FIG. 172a.—Myxedema, showing enlarged veins in case of Fig. 171b.

into utilizable metabolic constituents, or, as a modification of this idea, that the thyroid eliminates certain harmful elements from the blood. The blood in myxedema is poor in oxygen and the urine is of an increased toxicity. Both of these conditions are favorably modified, as is the myxedematous state, by the administration of thyroids. It now is determined that the thyroid gland is essential to life and to the proper neurotrophic balance. That it is essentially secretory is proved by its embryonal duct in man opening at the root of the tongue and its active connection with alimentary processes in the lower orders.

**Morbid Anatomy.**—The primal lesion of myxedema is located in the thyroid. In myxedematous idiocy the gland is either wanting, rudimentary, or atrophic. In acquired myxedema it is atrophic, yellowish white, and fibrous. At first there appears to be an embryonal vesicular

infiltration, with epithelial proliferation. Later, fibrous changes predominate, and finally complete sclerosis is presented. This practically constitutes a parenchymatous and interstitial thyroiditis. Murray, however, believes that atrophy of glandular tissue is the first step, due probably to the action of some toxic agent, and that the increase of fibrous tissue is a replacement-fibrosis. The cystic degeneration of cretinoid goiter reaches the same results by its destructive action upon the glandular parenchyma.

The subcutaneous cellular tissues are infiltrated with a gelatinous, mucinoid substance and the fatty parenchyma is much increased. Through-



Fig. 223b.—Shinings of hands of Fig. 222.

out the organism there is a tendency to fibrous proliferation, which especially affects glandular organs, as those of the skin and the kidneys. Mucin has also been found in the blood and in all the tissues of the body. The pituitary gland and the thymus are frequently enlarged, apparently for the purpose of compensation. The nervous system presents no changes that are uniform or significant. Central hyperemia and translocation of the nerve-cells have been noted by Rogowitch.

**Treatment.**—The treatment of myxedema furnishes one of the most brilliant chapters in medicine. It was observed that in animals and man myxedema did not follow partial ablation of the thyroid, and Schiff found that a portion of the gland subcutaneously or intraperitoneally implanted prevented the myxedematous state in animals subsequently thyroidectomized. Horsley suggested the same procedure in man, and it



was carried into effect by Kocher, Lannelongue, Bircher, and others with favorable results. When the thyroid graft was successful, myxedema was temporarily checked. Murray first used subcutaneous injections of thyroid extracts or emulsions with better and more persistent results, and a series of cures were reported by this method in the hands of numerous observers. Horvitz, of Copenhagen, followed shortly and independently by Mackenzie and Fox in England, and then by a host of physicians the world over, fed myxedematous patients with thyroids raw, partially cooked, or desiccated. The results were almost uniformly good. The procedure is perfectly justified not only by clinical results, but by the fact that in lower animals and in the human embryo the thyroid gland furnishes a secretion to the alimentary canal by a duct opening at the foramen cecum on the base of the tongue. The sheep's thyroid is habitually employed and is now an article of commerce in the form of various powders and tablets. The equivalent of one-half a sheep's thyroid may be administered daily, and if well tolerated, increased to a full thyroid or more. If prompt action is secured, it may be reduced, and if difficulties arise, it must be discontinued and resumed with caution. The exhibition of thyroids is followed in a day or two by a return of the temperature to the normal standard, which it may even exceed by a degree or two; the pulse becomes more rapid, the urine increases in volume, the pigmentary infiltration diminishes, the fatty deposits melt away, and the patient becomes more animated and cheerful. Four to eight weeks may show a marvelous change. The skin becomes softened and moist, the hair and nails are better nourished, and the normal state of the patient is shortly attained. To maintain the cure, thyroid feeding must be maintained. If the treatment is discontinued, the patient promptly relapses. The equivalent of one thyroid a week is usually sufficient for the purpose, and cases are now on record in which several years have been passed in apparent health and vigor under this régime.

The treatment is attended by considerable danger, and in some instances it has been followed by fatal results. Headaches, pains in the bowels, diarrhea, neuralgia, insomnia, malaise, and nervous excitement are some of the unpleasant symptoms which should promptly lead to a reduction of the dose or to the temporary interruption of the treatment. Some of these disadvantages are attributable to improperly prepared thyroids and ptomain poisoning. When the terminal cachexia of myxedema has appeared, this treatment is hazardous, but nothing else can possibly rescue the patient, and in some such cases it has done so. In children, especially in myxedematous idiots with rachitic deformities, thyroids tend to produce a softening of the bones that may greatly exaggerate the osseous distortions. Parker<sup>1</sup> and others have met with this complication, and have recommended that such cases be treated in bed, and the weight kept off the legs.

In acquired or operative myxedema thyroid feeding may be considered established as a curative treatment. In cases of myxedematous retardation its results appear equally brilliant. In sporadic cretinism it is capable of producing the most marvelous improvement, which is com-

<sup>1</sup> Brit. Med. Jour., July, 1896.

plete in proportion as it is adopted early in life. In endemic cretinism its effects are beneficial, but as yet its proper value is not established.

The action of thyroïdin, a chemical compound isolated by Baumann,<sup>1</sup> is practically the same as that of the various preparations of the gland, but is less likely to be attended by the accidents of ptomain poisoning. Against the theory that sometimes follows removal of the thyroid, it is less efficient than the thyroids themselves.

#### EXOPTHALMIC GOITER.

Exophthalmic goiter, otherwise known as *Graves' disease*, *B Basedow's disease*, *cardiosthyroid exophthalmos*, and *stomach exophthalmos*, presents, in most respects, a complete contrast to myxedema, but also shows some analogous features. We have considered myxedema as due to defective thyroïdation, and we may look upon Graves' disease as the manifestation of excessive or perverted thyroid activity or of both. The disease is manifest by the three so-called cardinal symptoms of rapid heart, enlarged thyroid, and prominent eyeballs. To this must be added a fine tremor, mental irritability, and muscular weakness, which are equally common. Further, nearly every function of organic life may be disturbed. Majari, in Italy, described the condition in 1802, and Parry, in England, in 1825. Graves taught it as a disease entity in 1835, and published it as such in 1843; meanwhile Basedow, in Germany, had independently written of it in 1840. This serves to explain the various names applied to it in different countries.

**Etiology.**—Graves' disease belongs to the *reproductive period* of life, being rare before puberty and after the menopause. Barrett<sup>2</sup> was able to collect only 12 cases in children below the age of fifteen, the youngest being four and a half. In children the disease is more acute and rapid in its course and development than in adults.<sup>3</sup> It affects females five or six times as frequently as males. A neuropathic heredity is commonly encountered. Pubescence in girls, menstrual difficulties, chlorosis, and all debilitating conditions act as predisponents. In exceptional instances Graves' disease seems to be associated with intranasal and intra-abdominal conditions. As *causæ* may be mentioned emotional and mental shocks, especially profound and protracted anxiety and grief, but frequently cases attributed to such causes can be traced back of them. In such instances the mental strain may serve to precipitate the more prominent symptoms of the disease. Pregnancy may seem to excite the disease. Occurring during its course, the disease is sometimes apparently modified for the better, but, on the other hand, may be aggravated. After delivery there is frequently distinct improvement. Graves' disease is often associated with other neuroses, such as chorea, hysteria, and epilepsy. It is observed sometimes with tabes, and the mental disturbance not infrequently reaches into the field of insanity. A family type<sup>4</sup> is sometimes encountered, affecting several or all offspring of parents who may show no abnormality. A special but rare association of

<sup>1</sup> Notkin, "Wien klin. Wochn.," Oct. 22, 1886.

<sup>2</sup> "Ann. de Méd.," July 10, 1892.

<sup>3</sup> Schickens, "Gazette Médic.," (Bassem), 1908, Nos. 1 and 2.

<sup>4</sup> Brover, "Chicago Med. Rec.," 1898; Holmes, "Phila. Med. Jour.," June 11, 1898.



Graves' disease is with tetany, scleroderma, myxedema, and acromegaly, all of which are closely allied through relation to the thyroid body and other endocrine glands.

Numerous theories as to its pathogenesis have been entertained. Graves considered it a disease of the heart. Marshall, Taylor, and Pierry attributed it to mechanical compression of the cervical vessels and nerves. After the experiments of Claude Bernard upon the cervical sympathetic, lesions of this portion of the nervous apparatus were believed to be at the bottom of exophthalmic goiter. Following Charcot, the disease was by many considered a pure neurosis similar to hysteria. At present there are two general theories in the field. One incriminates the nodulla, the other, the thyroid. Pointing to the *hæmorrhagic* origin of the disease is the association of cardiac, muscular, secretory, and thermal disturbances, to which, in various rare cases, palsies of cranial nerves, producing ophthalmoplegia, facial palsy, trigeminal neuralgia, and auditory disturbance are added. In certain cases bullæ hemorrhagicæ, atrophy of the pituitary bodies, and degeneration of the solitary bundles have been found, and the association with tuberculosis argues the same cause. Feline and Dardail, by experimental lesions of the pituitary bodies, have produced the major symptoms of exophthalmic goiter. As a rule, however, the bulb is not structurally affected, and the diverse conditions cited are as likely to be effect as cause.

Following Johnston there are many who believe that overactivity of the thyroid gland, causing a *hyperthyroidism*, explains the genesis of exophthalmic goiter. The principal facts supporting this point of view are: (1) The usual changes and enlargement of the thyroid; (2) the improvement and cures resulting upon removal of a portion of the gland; (3) the symptoms of exophthalmic goiter induced by overdoes of thyroids in myxedematous patients and normal individuals; (4) the cases in which exophthalmic goiter has eventuated in myxedema through degenerative changes in the gland, and (5) the almost absolute contrast between myxedema and Graves' disease.

Horsley, who gave most constant and careful attention to this subject for many years, and to whose investigation we owe much of our present knowledge regarding the functions of the thyroid body, insisted upon the changes in the gland and its secretion. He declared that "exophthalmic goiter in its various degrees results from perversion of the function of the thyroid gland."<sup>1</sup> It has been found by many observers that the use of thyroids in Graves' disease often intensifies all the symptoms, but there are certain cases in which they seem to cause improvement. Is it not reasonable to suppose that when the gland is simply or mainly overacting their administration increases the hyperthyroidism, but may benefit the cases that by perversion of thyroid function are not supplied with a normal secretion? Cunningham,<sup>2</sup> on experimental grounds, reaches with Gley the conclusion that the majority of symptoms in Graves' disease may be plausibly explained by the hypothesis of deficient thyroid activity. This Kocher, on very logical grounds, entirely denies.<sup>3</sup> The question of the relation of thyroid action to the functions

<sup>1</sup> *Brit. Med. Jour.*, Dec. 5, 1896.

<sup>2</sup> *Jour. of Experimental Med.*, 1898.

<sup>3</sup> *Brit. Med. Jour.*, June 2, 1900.

of the nervous system has been already outlined in the description of myxoedema (see p. 324).

Accepting the thyroid theory, we are still confronted with the initial question as to why the thyroid function is primarily disturbed. Some have attributed this to infection setting up a thyroiditis, and point to the frequent history of antecedent infectious fevers, etc. Others accuse the neuropathic heredity or constitution. The real cause still escapes us. The intimate relation of thyroid and uterine functions (see p. 324) and the usual occurrence of exophthalmic goiter during reproductive life are of interest.

**Morbid Anatomy.**—The autopsical findings in exophthalmic goiter are inconsistent and variable. The heart is often dilated; the sometimes thickened walls may present fatty degeneration. The valves are diseased only as accidental concomitants. The thyroid body may present any goitrous variation from simple congestion to the most destructive lesions. Usually the lobes are unequally enlarged, firm to the touch, and present a reddish, pulpy aspect. Colloid degeneration in places is not uncommon and may result in cyst formation. The vessels are thickened, dilated, and, in chronic cases, atheromatous. There is a proliferation of connective tissue throughout the gland which may reach a sclerotic degree. The acini of the gland become dilated; the colloid material disappears, leaving a granular debris; the secretion becomes thin and watery, the epithelium more or less disintegrated. The thymus is often persistent and enlarged, showing increased vascularity and an attempt at compensatory or perhaps at antagonistic activity. Capelle found it enlarged in 80 per cent. of 60 fatal cases, and Orel and Mackenzie assert that it was enlarged in all cases examined by them. Carl Hart<sup>1</sup> even distinguishes a pure thymus form of Graves' disease, but Kocher<sup>2</sup> seems to deny the essential thymus variety and does not consider the surgical removal of the thymus as definitely indicated. The orbital cavity is often normal, but as frequently presents some fatty proliferation, and almost always the evidence of a continuous retrobulbar congestion.

On the part of the nervous system the alterations in the cervical sympathetic ganglia, described in early accounts, are not found. Changes attributable only to the cachectic state are detected. In the bulb and spinal cord vascular degeneration has been encountered, with occasional small hemorrhages and dilatations. Atrophy of the testiform bodies and of the solitary bundles has been noted once or twice, as has atrophy of the ascending root of the trifacial. The associated lesions of tubes are more frequent.

The muscles, according to Askany,<sup>3</sup> show fatty infiltration, usually distinct to the naked eye. The fat globules are seen microscopically in long rows in the muscle-fibers, which also show increased, sometimes degenerated nuclei.

**Symptoms.**—The onset of exophthalmic goiter is frequently insidious, and the patient can scarcely say when it began. In other in-

<sup>1</sup> "Archiv. f. klin. Chirurgie," Bd. 104, p. 347.      <sup>2</sup> *Ibid.*, Bd. 100, p. 924.

<sup>3</sup> "Deutsch. Arch. f. klin. Med.," Band 61.



stances the major symptoms appear within a few days or even within a few hours, following some exciting shock, but it is always allowable to suppose that unobserved minor manifestations had been previously present. Such fact can frequently be elicited by judicious inquiry. Cardiac palpitation, nervousness, irritability, and unaccountable fatigue are often felt for months before the more prominent symptoms are declared.

**Cardiovascular Features.**—The *cardiac disturbance* usually is the first of the triad of cardinal symptoms to appear. The pulse becomes rapid, and ordinarily reaches 120 a minute, unless the patient is reclining and at perfect rest, when it may drop to 80, but never reaches normal during the activity of the disease. A rate of 160 or even 200 is not very rare. Of the three major symptoms a rapid pulse may alone exist, and, taken with several other and minor symptoms, may suffice for a diagnosis. It is the only essential symptom. Ordinarily regular, the rhythm of the heart-beat in some cases is greatly disturbed. The heart acts in the most tumultuous, irregular manner, and its incompetence is shown by vertigo, cyanosis, and precordial distress. Palpitation and cardiac throbbing are experienced by most patients, and may be occasioned by the slightest physical or mental distress. At such times the breast, neck, and face are frequently suffused, and hot waves, with violent blushes, may sweep over the upper part of the chest, neck, and face. At first, and sometimes throughout the disease, the stethoscope reveals nothing but the tachycardia and violent systole. Functional systolic bruits and anemic murmurs are not infrequent. Organic valvular disease is practically an accidental complication. An apparent hypertrophy is really due to dilatation and to the enlarged area over which the apex-beat extends by its increased violence. In late cachectic stages the dilatation, with degeneration in the heart-muscle, becomes physically apparent and threateningly prominent. Grosso<sup>1</sup> finds that dilatation is always present during attacks of tachycardia, that there is an intimate relation between the amount of dilatation and the general asthenia, and that intense but transitory changes in the *consistency and shape* of the heart are marked features of the disease.

The entire circulatory system is affected. This is most manifest in the aortic branches, especially in the cervical arteries, and is seen in the temporal and retinal arteries and veins. It is less well marked in the extremities, but Gerhardt<sup>2</sup> has noted it in the palmar arches and in the crurals. He also calls attention to the presence of capillary pulsation in the spleen and liver and kidneys. In cases of long standing, vascular dilatation is produced and the veins themselves become fibrinous and arterIALIZED. The blood shows mainly the alterations of a simple anemia, but without increase of the total number of white cells (the lymphocytes frequently are increased even to 60 or 70 per cent., and 30 per cent. is very common (Koeber).

The goiter may appear at any period of the disease or may never develop. In some instances it is of insidious growth, and the patient's attention is only incidentally called to it by tightness of the ordinary

<sup>1</sup> "Rev. crit. di Clin. Med.," Jan. 2, 1902.

<sup>2</sup> "Centralblatt f. Chirurgie," Sept. 5, 1896.

neckwear. In other instances it is of sudden development, or may advance in size at intervals, or advance and recede repeatedly. In any case it rarely attains very large proportions, and practically never interferes mechanically with respiration. It is usually asymmetrical, the right lobe commonly being most affected. The swelling may be confined to one lobe, usually the right, or it may involve the isthmus alone. The tumor usually offers considerable resistance to palpation, and a pulsatile thrill is frequently to be detected. In some instances nodules, indicative of parenchymatous changes, can be made out. Auscultation over



FIG. 225C.—Enlarged  
Thyroid Gland.

the enlarged thyroid readily detects in most cases a bruit synchronous with the pulse, and the tumor can often be seen to expand with every systolic impulse. By manual compression it may often be greatly reduced in size for the moment, and it has a tendency to fluctuate in volume during the progress of the disease, increasing in size upon the occasion of any physical or mental excitement. In late cases, through interstitial thickening or cystic degeneration, it may give a *sclerous* or *fluctuant* feeling to the touch, and does not then recede upon the cessation of the malady. It may even become markedly atrophic, and then myxodema gradually develops. In a few cases a goitrous condition may have been present for many years and the glandular tumor may have attained

large proportions before hyperthyroidism symptoms are superadded.

**Ocular Conditions.**—With the appearance of *exophthalmos*, which usually promptly follows the goitrous enlargement, the famous triad of symptoms is complete. It may appear before the goiter or in cases that never show thyroid enlargement. Both eyes commonly are affected, but at first usually in unequal degree, and exceptionally but one is prominent. This occurs usually in cases in which only one lobe of the thyroid is enlarged, and commonly on the same side.<sup>1</sup> The prominent eyeballs with wide-open lids give an expression of excited fierceness and fright strangely mingled, and quite disconcerting to strangers. The ocular protrusion varies greatly in amount in different patients, but in extreme cases has caused actual dislocation of the eyeball. Unless it exceed a moderate amount it occasions no inconvenience and may even escape the patient's attention. In a more pronounced degree the exophthalmos induces some difficulty in ocular movements and causes ocular fatigue. When exophthalmos is well marked a distinct bruit has been heard by placing a stethoscope on the closed lids.<sup>2</sup> The cornea may also become inflamed from inadequate protection, especially at night and in the wind. There is frequently an increased *lacrimation*, which may occur independently of the exophthalmos, and later the secretion of tears may be defective. *Conjunctivitis* and *keratitis* may arise as complications, and *perforation* by ulceration has been known.

<sup>1</sup> Fridenberg, "Med. Record," July 18, 1903.

<sup>2</sup> Sanger Brown, personal communication.



The lids are usually retracted and often show peculiar and important motor difficulties. Winking is infrequent, but in occasional instances is rapidly repeated at momentary intervals. Von Graefe noted that in looking down the upper lid did not correspondingly follow the movement of the ball as in health. This sign is not related to the amount of exophthalmos, as it is sometimes found in normal individuals, and may be absent in marked cases of Graves' disease. Stellingma called attention to the usual widening of the palpebral fissure and the incomplete closure of the lids even when the patient thinks the eyes are firmly closed. In a few cases Joffroy has noted a similar inactivity of the lower lid and of the frontalis in looking upward, voluntary control of the latter muscle remaining perfect. The outline of the palpebral fissure is often more or less angular, losing its smooth curves. Miéens has called attention to a difficulty of convergence that is commonly present. Kocher<sup>1</sup> considers a sudden retraction of the upper lid when the patient is directed to look steadily at the examiner or upward a very early and persistent symptom. In exceptional cases an external ophthalmoplegia has been seen. Paralysis of the frontalis or of the entire facial nerve or involvement of the sensory and motor portions of the trifacial has been noted. Pigmentation of the lids is often excessive.<sup>2</sup>

Vision is usually intact and the pupils are generally normal, but nyctopia may develop on extreme pressure from exophthalmos. According to Sauton and Rathery,<sup>3</sup> the pupils may be affected in four ways: (1) dilatation, which is relatively common; (2) contraction, which is less frequent; (3) inequalities, which are rare; and (4) dilatation, later changing to contraction, which is extremely uncommon. The eye-grounds show no abnormality aside from retinal congestion and dilated, sometimes pulsating, vessels. Photophobia and brilliant nyctopia are attributable to the retinal congestion, and hallucinations of sight may have the same origin.

**Motor Conditions.**—Besides the motor disturbances of the eye and its appendages, previously indicated, the entire musculature is affected with such marked weakness that Charcot was led to describe a Basedowian paraplegia. Muscular adhesion is often an early symptom. The reflexes may be diminished and the legs may suddenly give way, causing the patients to fall heavily. Of similar origin are the shallow respiratory excursions and diminished expansion of the chest. Cramps, contractures,



FIG. 226.—Exophthalmos grave: (1) Profile view showing the protrusion of the eye; (2) Frontal view showing the protrusion of the eye and the retraction of the upper lid.

<sup>1</sup> "Ber. Med. Jour.," June 2, 1906. <sup>2</sup> Jellinek, "Wiener Med. Woch.," 1904, Nr. 42.

<sup>3</sup> "L'Œophthalme," 1908, No. 7.

fleeting tetanoid conditions, and even epileptiform crises have been noted. The occasional association of chorea, epilepsy, and hysteria has already been mentioned, and must not be confounded with motor disturbances due to Graves' disease. Tetany has been seen by the writer in one case in the terminal period of the disease.

An almost constant symptom in Graves' disease is a *fin de frêne* which is as significant as any one of the cardinal triad. It is of variable intensity, may appear only at intervals, and be limited to the head and upper extremities. By placing the finger-tips on the head when the patient is sitting or standing, the examiner will be aware of a vibration, and this may be demonstrated by placing a long feather in the patient's hat. It usually is easily seen in the extended hands, especially if the fingers be spread widely, or it may be then felt by grasping the patient's wrist. In some cases the entire trunk is affected, and in standing, the tremor may be present in the lower extremities. It is sometimes distressing to the patient, but often occasions no complaint, and must be intelligently sought. The rapidity of the tremor is from eight to ten oscillations a second.

**Secretory and Vasomotor Features.**—With the hot waves and morbid flushing that have already been noted there is often profuse *perspiration*, which may be widely distributed, limited to one side of the body, or most abundant on the hands and feet. In some cases it necessitates frequent changes of underclothing. Usually it is attended by a feeling of almost unbearable heat, and these patients often seek cool rooms even in winter and find all bed-covering intolerable. The bodily *temperature* may be quite normal, but in rare instances an elevation of from one to five degrees has been noted, and in the terminal cachexia of fatal cases hyperpyrexia is commonly encountered. *Hemorrhages* from the uterus, nose, gums, and lips are not infrequent.<sup>1</sup> *Polyuria*, especially periodical polyuria, is a common phenomenon, and *glycosuria* is sometimes encountered. *Albuminuria*, however, is the rule at some period of the disease. It is usually intermittent and appears when the other manifestations of the disease are exaggerated, as if due to the hypervascularity of the medulla. *Edema* is common, but peculiar and usually circumscribed; it may affect the upper eyelids and has a preference for the outer aspect of the legs, the thighs, and the abdomen. The infiltration is not boggy and does not pit on pressure, but reminds one of the resiliency of myxedematous swelling. In rare instances it may become generalized and does so in those cases that eventually develop myxedema.

**Mental Disturbances.**—From the beginning, and often for a long period antecedent to the appearance of cardiac symptoms, the subjects of Graves' disease present a considerable *mental restlessness*. There is an indefinable and tormenting agitation, marked by mental and motor restlessness and an imperative and impulsive tendency to be doing. Their emotions are too readily excited, and they are unusually impressionable and irritable, reacting in an exaggerated manner to all the incidents of daily life. In more pronounced cases they become volatile

<sup>1</sup> B. Popoff, "Neural Centralid," April, 1900.



and manifest the greatest mobility of ideas, without persistent concentration or logical order. Their affections are likely to undergo modifications, and they become irascible, fault-finding, inconsiderate, ingenuous, and hard to live with. In some instances this disturbance of mentation carries them over the border into active mania, marked, perhaps, by delusions of fear, due to the cardiac symptoms or the sensations of heat. Insomnia is often added, and the fitful sleep is disturbed by horrifying dreams that are likely to be projected into the waking moments and woven into delusions which are usually unsystematized, and constantly changing, furnishing the analogue of the motor restlessness. Hallucinations of sight and hearing are not uncommon.

The mental perturbation only rarely reaches the degree of actual mania, and then is, perhaps, equally dependent upon numerous other causes acting in a neurotic individual. But a condition of *obsessive mental stimulation* is characteristic of the anxiety, and as important an index as any of the cardinal triad.

The skin is often marked by *obsessive pigmentation*, which may be distributed in irregular plaques over the body, face, and limbs, or in rare instances produces *ash-leaf-like* markings on the trunk. In certain cases it much resembles the bronzing of Addison's disease, but usually spares the mucous surfaces, though not always. Vitiligo, scleroderma, and various cutaneous eruptions are sometimes encountered, and the hair may become thin. The sensation of heat is usually associated with a *diminished resistance to electricity*, which may be reduced to a third or a fifth of the normal. This is possibly related to the excessive perspiration, but does not obtain in other diseases in which sweating is equally great.

Digestive disturbances are numerous and not marked by anatomical lesions. Anorexia, voracious appetite, vomiting, diarrhea, dysentery, and icterus may be encountered in different patients, or many of them at various times in the same patient. The *diarrheas* are particularly important, as they rapidly reduce the patient's strength and tend to hasten the appearance of cachexia and a fatal termination by exhaustion. The frequent painless stools are made up of undigested food and an abundance of watery mucus, sometimes liberally streaked with blood. The defects in nutrition are always pronounced and a *loss of weight* from the first is the almost exceptionless rule. It may occur intermittently, the patient losing eight or ten pounds in a week and gradually regaining it. Hurland<sup>1</sup> is inclined to look upon such loss of weight, and especially upon its intermittent occurrence, as of diagnostic importance. It may occur independently of the diarrhea, sweating, polyuria, or any other physical drain, and in the face of an active bulimia.

Respiratory disturbances are not uncommon. They are frequently secondary to the cardiac disorder. Dyspnea, asthmatic attacks, pulmonary congestions, bronchitis, and a persistent cough, without stethoscopic symptoms, may be presented. The dyspnea is the same as

<sup>1</sup> "Jour. de Méd." Feb. 10, 1885.

that in myxedema, and appears to be due to the atrophic condition (Horsley). Shallowness of respiration and inefficient thoracic expansion are related and proportionate to the muscular asthenia.

The genital functions are usually modified. Increased sexual appetite has been noted in men and women in the early periods of the disease. Later, as general asthenia develops, it is correspondingly reduced. Amenorrhea is present, or the tendency is well marked, and leukorrhœas are abundant.

The following table of symptoms and their relative frequency is based on Paresce's statistics,<sup>1</sup> comprising fifty-eight cases, of which forty-two were women. It must be borne in mind that nearly all the symptoms of Graves' disease are intermittent, and any tabulation, to be satisfactory, should cover the entire course of the disease and embrace a very large number of cases:

TABLE SHOWING RELATIVE FREQUENCY OF SYMPTOMS IN GRAVES' DISEASE, BASED ON FIFTY-EIGHT OBSERVATIONS.

Neurotic family history . . . . .	29	Sensations of warmth . . . . .	24
Tachycardia . . . . .	57	Vertigo . . . . .	23
Nervous irritability and restlessness . . . . .	17	Insomnia . . . . .	21
Characteristic tremor . . . . .	52	Stefling's symptom . . . . .	20
Palpitation and vascular disturbance . . . . .	50	Tachycardia, prior, exophthalmos, and tremor combined . . . . .	18
Goiter . . . . .	48	Polypsi . . . . .	17
Increased perspiration . . . . .	34	Cardiac dilatation . . . . .	15
Anæmia . . . . .	34	Irritable cough . . . . .	10
Severe headaches . . . . .	22	Græfe's symptom . . . . .	9
Exophthalmos . . . . .	28	Müller's symptom . . . . .	9
Severe diarrhea . . . . .	2	Valvular heart disease . . . . .	5

**Course and Progress.**—The great diversity encountered among cases of Graves' disease makes it difficult to outline its clinical course. It may be of apparently sudden or of insidious onset, and may show any combination of the long list of symptoms that have been detailed above. It may run its course to a favorable termination in three months or may take six years, or continue throughout or terminate life. The great majority of cases are of protracted duration. Early recoveries and fatalities are alike exceptional, and when all other symptoms have subsided, prominent eyes and a moderate goiter may remain to mark the passage of the disease. The appearance of active diarrhea, of polyuria, intense albuminuria, and rapid wasting darkens the prognosis. Extreme myxædema, great cardiac dilatation, and a failing circulation have the same import. Mental disturbance is not of itself of bad prognosis, which depends rather on failing bodily conditions. The cardiac symptoms are usually the first to appear, and afford the best criterion as to the progress of the disease. Their improvement is the only unqualified sign of gain. A state of nervous susceptibility is usually left after recovery that may be compromised by the slightest shock.

**Diagnosis.**—In the presence of the Basedowian triad diagnosis can not fail, but in the abortive forms it requires a careful and thorough

<sup>1</sup>"Deut. Zeit. f. Nervenk.," Bd. vi, 8, 21.



investigation and an experienced judgment to recognize the disease. Tachycardia, with irritability, sweats, insomnia, emaciation, and perhaps a slight rise in temperature may mimic *phthisis* very closely, and if an irritable cough is added, the mask is almost impenetrable, especially as some pulmonary dullness, due to the congestive condition, may be present. The mental attitude is of some assistance, and should the tremor be observed or some slight throat turgescence be made out, the diagnosis may be effected. Some of these *formes frustes* are put down as *neurasthenia*, in view of the asthenic condition so commonly present. It requires the presence of eye, thyroid, cardiac, or tremor symptoms to make the diagnosis positive. Every case of Graves' disease is at the same time myasthenic, and may also be neurosthenic.

**Treatment.**—In the management of this disease, whatever line of treatment may be added, it is of the utmost importance to secure rest and quiet. With complete rest, as by the Weir Mitchell course, some cases



FIG. 225.—Partial recovery. Some exophthalmos, pigmentation of skin, and generalized hyperkeratosis.

make immediate and substantial progress. If this is not available, it should be approached as nearly as the circumstances of the patient permit. In the asthenic, irritable state, a conservation of energy is strongly indicated. *Digitalis*, *strychnine*, and *strychnin* have a favorable action on the heart in some cases, and strychnin in full doses is occasionally of benefit to the general nervous condition. Sedatives such as *bromide* and *belladonna* have not yielded the writer good results in any instance, but chloral is often valuable for the insomnia. *Iodo* must be used with caution even to combat the anemia so commonly present, as it frequently disturbs the intestinal tract and augments the vascular storm. *Hydrotherapy* and *massage* are of benefit in some cases. Electricity locally has strong myotonic, some favoring fibrillar, others galvanic. A moderate faradic current through the root of the neck, causing all the anterior cervical muscles to contract about the thyroid, mechanically diminishes it in size. This may last a few minutes or for a few hours after the current is stopped if the patient is not excited in any way. An uninterrupted galvanic current may produce a similar

result, and passed through the temples occasionally causes a recession of the eyeballs, but the effect is very fleeting and does not influence the tachycardia. Electricity should be used daily, or several times a day. Mechanical compression of the thyroid by bandaging is insupportable.

*Injections of caustic agents*, such as tincture of iodine, into the substance of the gland, at one time much in vogue, are now generally abandoned, as they are of doubtful value and are attended by considerable danger to life. Injections of boiling water have been used with alleged immediate improvement, but all such measures must be inexact, as they depend upon local necrosis for their effect. In long-standing cases ablation of a portion of the gland has produced good results in the hands of various operators, but it has a considerable mortality—about 12 per cent. Schulz<sup>1</sup> reports 14 cases with 12 cures, but Senger<sup>2</sup> cites a case in which all symptoms were aggravated. Cystic portions may be removed, but caution must be exercised to leave a fair amount of normal gland. The same effect is obtained by exposing the gland by a median incision and securing it in the wound (the *exothyropey* of Jaboulay), but even this comparatively simple operation has been followed by death. Thus operated, the thyroid shrinks in size and the symptoms decrease. The wound is closed at a subsequent period. Surgeries are meeting with better results from partial thyroidectomy, especially in the less advanced cases, now that the parathyroids have been recognized. When they can be isolated and spared, the operation is much less objectionable. It must receive careful consideration in every case. The mortality is still considerable. Tabulated results show this to be about 10.5 per cent. Thus: Moses, 3.5 per cent.; Starr, 12.1 per cent.; Kinnient, 3.9 per cent.; Rich, 13.1 per cent.; Lengo, 13.9 per cent. In the hands of Mayo and Kocher the mortality has gone down to less than 5 per cent. Jounesse<sup>3</sup> and others have claimed improvement from bilateral ablation of the cervical sympathetic. The operation is a serious one, not without bad results,<sup>4</sup> and should be condemned. The detection of an enlarged thymus gland makes prominent the probability of serious or fatal conditions arising under surgical proceeding.

*Thyroids* have been used. Ordinarily, they intensify the symptoms. In a minority of instances the goiter is reduced in size quite promptly, but the pulse is usually not improved, and the nervousness and tremor are generally accentuated. They may be carefully tried experimentally. A number of cases have been benefited by the use of thymus feeding. Owen<sup>5</sup> has collected about 20 cases in which its effect was mainly beneficial. Mackenzie,<sup>6</sup> in an equal number of cases, found it of little or no value. Owen called attention to the probable physiological antagonism between thymus and thyroid action. The persistence or reappearance of the thymus in these cases, often noted, among others by Hektoen,<sup>7</sup> may have the same significance. Some encouraging results have been obtained by feeding patients on the milk

<sup>1</sup> "Berliner Klinik," June, 1887.

<sup>2</sup> "Munch. med. Wochensh.," April 6, 1887.

<sup>3</sup> "Centralbl. f. Chir.," Jan. 16, 1897. <sup>4</sup> "Archiv. f. Rev. de Neurol.," Aug., 1900.

<sup>5</sup> "Brish Med. Jour.," Oct. 10, 1895. <sup>6</sup> "American Journal," Feb., 1897.

<sup>7</sup> "International Med. Mag.," Sept., 1893.



of thyroidectomized goats or using a serum obtained from the blood of thyroidectomized animals. Several hemic products from such animals are being put upon the market, but their value is as yet undetermined. Ovarian preparations and adrenalin have also been used with uncertain and unreliable effects, but Hoppe<sup>1</sup> strongly advocates the use of corpus luteum and reports favorable results well maintained by its continuous administration.

The numerous *complications* must be met as they arise. Atropin for the sweats, bismuth and opium for the diarrhoea, careful dieting for the gastric and intestinal troubles and a general tonic and upbuilding régime. Thompson, of New York, insists on a milk diet.

#### SCLERODERMA.

Scleroderma is a glandular trophoneurosis presenting an induration and atrophy of the skin, or atrophy without preceding induration. It occurs (1) in a generalized form, but affecting the extremities and face principally, and (2) in a circumscribed form, marked by isolated plaques and stripes. These may be confined to one side or be roughly symmetrical in distribution, and are often limited to the cutaneous extent of certain nerve-trunks or branches. This led Jonathan Hutchinson<sup>2</sup> to use the term *herpetiform morpha*. The disease is also known as the hide-bound disease, *scleroma adultorum*, *sclerosis*, *chlorosis*, *morpha*, and Addison's keloid. When the fingers are affected, the French employ the term "*sclérodactylie*." It is a rare disease, but not so uncommon as most writers insist, and if generally recognized would soon present a generous material.

**Etiology.** The female sex furnishes about three-fourths of all cases. Generalized scleroderma is most common in adults, but may be encountered between ten and twenty years of age, and the discrete form begins perhaps more commonly before twenty-five than later in life. It has been attributed to various infectious fevers, to traumatism, exposure to cold, rheumatism, and to pretty much every incident of human life. It probably has a relation to disease of the ductless glands, and particularly to disorder of the thyroid. A neuropathic constitution is of such striking frequency in these cases that it cannot be overlooked. Spinal cord and cerebral lesions are sometimes associated, and in the generalized form Raymond's disease is



FIG. 226.—Generalized scleroderma, showing face, sclerodactylia and atrophied limbs (Grunz).

<sup>1</sup> *Jour. Nervous and Mental Dis.*, April, 1918, p. 254.

<sup>2</sup> *Brit. Med. Jour.*, June 1, 1895.

a frequent concomitant. I have seen it associated with a progressive myopathy. Insanity is sometimes present, especially melancholia.

**Symptoms.**—In the generalized form of scleroderma at first there is irritation followed by induration and thickening that may be red and suffused by vascular stasis and present blebs and bullae. Eruptions urticaria and dermatographia are commonly easily demonstrated.<sup>1</sup> On the broad surfaces of the trunk the skin feels stiffened and heavy. It is found to be bound down to the underlying parts, giving rise to the hide-bound descriptive title. Atrophy occurs later. All the dermal structures become thinned and present a cicatricial appearance, and may be glistening white. The hands and face are especially affected. The fingers are reduced to their slightest proportions, and the bones, covered by the atrophic, glistening skin, which fixes the joints and limits motion, are lessened in size. Raynaud's disorder is often present. Over the facial bones the parchment-like integuments are tightly drawn in a motionless, uncrinkled mask. The thinned lids can scarcely be closed over the sunken eyeballs, which consequently appear unduly large. The nose is thin and looks ready to split its way through the skin. The lips are papery and often retracted, exposing the teeth. The shortening of the skin from the jaw to the clavicles may even interfere with movement and with deglutition. Every bony prominence, as about the orbits, cheeks, and chin, is sharply defined. The epithelial structures are everywhere redened, and nails and hairs are of defective growth. Sensibility is not much disturbed, as a rule, but hypersensitiveness may be encountered.

In the discrete form a local thickening may gradually invade the surrounding parts, but is limited by rather sharp borders and may be attended by herpetic eruptions. Later it becomes atrophic. In other cases a white atrophic spot appears and gradually extends over the course of the supplying nerve. It may thus produce miter-like bands on the trunk and stripes down the limbs. The most common locations are the face, chest, and lower extremities. The outlines of these morbid tracts and plaques sometimes forcibly remind one of the cutaneous areas of the spinal segments.

Up to the time atrophic changes occur, the disease may recede and the normal condition be spontaneously established. Several years are usually consumed in the development of the disease.

Anatomically, the disease is marked by a fibrosis attended or preceded by vascular changes. The nerves and all structures within the atrophic area equally share the fibrous proliferation.

**Treatment.**—Tonics, electricity, hydrotherapy, massage, and general reconstructive measures have been found of some benefit in a few cases. Of late, Lustgarten, Sachs, Bramwell, and others have reported great benefit from thyroid feeding, which, even in advanced cases, caused immediate and gratifying changes for the better. In recent cases it appears likely to produce a cure. Whether a relapse will follow discontinuance of the remedy is not now certain.

<sup>1</sup> Lassar, "Nouv. Icon. de la Sclérot.", 1900.



**Facial hemiatrophy** furnishes a striking instance of localized scleroderma. To it is frequently added manifestations of organic disease of the fifth cranial nerve and the cervical sympathetic. In this rare deformity the wasting, commonly limited to the distribution of the trifacial, is usually most intense in the field of the middle and inferior divisions. The alleged causes in numerous instances, such as blows on the head and face, infectious fevers, exposure to cold, facial erysipelas, osteitis of the jaws, etc., are capable of seriously influencing the nerve, but their specific influence is questionable. In several cases histological changes in the trunk, ganglion, or branches have been demonstrated, and atrophy has followed division of the root of the trigeminal in man and animals. *Touche*<sup>1</sup> has reported lesions in the pons affecting the root



FIG. 236.—Two cases of facial hemiatrophy.

of the fifth nerve, and in another case the *sole* lesion was in cortex and pia of the operculum. *Jacquet* has demonstrated a lesion of the third cervical sympathetic ganglion, and many assume that the sympathetic is alone at fault. On the other hand, *Gruber*<sup>2</sup> reports a marked case without the slightest discoverable change in the nervous system. Facial hemiatrophy may occur in syphilitic cases as a local expression of luetic action; it may be a fractional part of syringomyelia or of any process that affects the trigeminal. In some instances similar wasting is present in the arm and shoulder, on the entire side of the body or on both sides of head and body. In certain rare instances other plaques or stripes of scleroderma are found widely distributed in the same patient (*Cassirer*).

Clinically, it may begin in a widening sclerodermic patch on the side of the face, but more commonly the entire half of the face gradually diminishes. The loss affects both dermal and osseous structures and less markedly the muscles, which may escape entirely. The opposite side of the face may finally become involved, though this is rare. The disease develops usually before adult life, but may appear at any age. It produces a most notable difference of appearance on the two sides.

<sup>1</sup>"*Neurol. Centralbl.*," June 16, 1914.

<sup>2</sup>"*Rev. neurologique*," 1912.

The atrophic half lacks the proportions of the sound side in every particular. The condition is usually most marked in the lower portion of the face, gradually lessening upward, so that the brow may show almost no discrepancy on the two sides. The skin



FIG. 2256.—Case of facial hemiatrophy (after Cooper).

is thinned notably, sometimes to a half or quarter of its proper thickness; the muscles are sometimes reduced in size and strength; the lower jaw may be a third smaller on the affected side. The teeth are often lost. As the skin is closely applied to the muscles and the bony conformation, a cadaverous appearance is presented that may be strikingly at variance with the plump, healthy side, and is sometimes sharply marked by a furrow at the middle line of the brow and chin. The nose, chin, and mouth deviate to the affected side. While the orbital and palpebral structures are frequently wasted, the eyeball is affected only in rare cases, but has been observed wasted and even destroyed. The disease is progressive for years, but may come to a standstill at any time, and again advance. It seems to be unmodified by treatment, but the continuous use

of thyroids may be tried with some prospect of retarding the progress of the wasting. Paraffin has been injected in a number of instances to restore the contour of the face purely for cosmetic effect, but the danger of producing a paraffin tumor makes this procedure ill advised.

### TETANY.

*Tetany*, or *tétanos*, is marked by peculiar tonic, bilateral, paroxysmal, muscular contractions, commencing in and usually confined to the extremities, and presents an increased mechanical and electrical excitability of the nerves. It may occur in epidemics, is endemic in many places, and commonly develops on a background of malnutrition.

**Etiology.**—It occurs most frequently before twenty and practically never after fifty years of age. Both sexes are affected, males more frequently before adult life, females more commonly thereafter. In Paris, Berlin, Prague, Vienna, and in Syria it may be considered endemic, and epidemics of it have been noted in those places, and in schools and garrisons. In the neurological clinics of Berlin <sup>1</sup> tetany furnishes one per cent. of all cases. In Vienna seven-tenths of one per cent. It appears most commonly from January to May. In America it is comparatively rare outside of quarters in large cities largely populated by foreigners. *Predisposing causes* include nearly every variety of deprived nutrition. Sarbo<sup>2</sup> from an extensive review of the causes of tetany, asserts that

<sup>1</sup> Lasker, "Boston Med. and Surg. Jour.," Nov. 19, 1890.

<sup>2</sup> "Deut. Zeit. f. Nervenheilk.," 1896.



impaired nutrition is the only common factor. Of most importance are chronic gastro-intestinal disorders, especially gastric dilatation, with hypermobility, fermentative diarrhea, and chronic constipation. Rickets is a very common accompaniment in children. Cassel<sup>1</sup> found it in 58 out of 60 cases. Tetany may follow acute infections and septic processes, or appear during pregnancy or lactation. Excision of the thyroid has been followed by tetany in a considerable proportion of cases. Billroth and Wölfler reported 19 cases of tetany in a total of 123 thyroidectomies. There is a certain relation between tetany and myxoedema, which may occur in a given case. If about one-fifth of the thyroid is spared, tetany does not appear, according to von Eisberg. Murray insists that it does not occur if the parathyroid is spared. As exciting causes of the paroxysmal attacks, exposure to cold and emotional disturbances are often active, as are overexertion, exhaustion, acute diarrhea, and vomiting. The administration of ergot, chloroform, and alcohol has caused them to appear, and they may be induced by mechanical irritation of the nerve-trunks and blood-vessels, in the manner to be described later.

The epidemic, endemic, seasonal, remissive, and toxic features of the disease point very strongly to the activity of some zymotic agent, but as yet it has escaped detection. Ohlo<sup>2</sup> suggests that it may only be active in the presence of some special form of perverted digestion. The vulnerable portion of the nervous system appears to be the spinal and bulbar gray and the peripheral nerves. The relation of the parathyroid bodies to the tetany of operative myxoedema and that of parathyroid-ectomized animals points to the probable participation of these glands in the development of ordinary tetany. The parathyroid bodies have been found variously diseased in many cases of tetany. Beside chromophilic and cytological abnormalities, hemorrhages, cysts, leukemic infiltration, tuberculosis, bacterial emboli, and adenomatous changes have been seen (Erdheim). Yunase found hemorrhages in 33 out of 89 cases in children.<sup>3</sup> McCallum and Voegtlin<sup>4</sup> have demonstrated that the parathyroids practically control the calcium metabolism of the body and that ablation of the parathyroids is followed by a reduction of calcium salts in the blood, and that the administration of calcium salts prevents or reduces the tetany. It is noticeable that conditions requiring calcium metabolism are usually associated with the tetany. Thus dentition, rachitis, pregnancy, lactation, and protracted digestive disorders are the common concomitants. Hence the value of a milk diet which is rich in calcium in these cases. Morbid anatomical changes in the nervous system are not constant, though suggestive. Cloudiness and swellings in the anterior horns have been noted by Wiess and by Barone and Cervasato. Peters,<sup>5</sup> in a recent communication, claims to have found in 7 cases an acute inflammation of the extradural connective tissue among the extradural blood-vessels and fat, constituting a pachy-

<sup>1</sup> "Deut. med. Wochs.," Jan. 28, 1907.

<sup>2</sup> "Munch. med. Woch.," Nov. 10, 1906.

<sup>3</sup> "Jahrb. f. Kinderheilk.," 1907.

<sup>4</sup> "Johns Hopkins Hosp. Bull.," March, 1908.

<sup>5</sup> "Deutsch. Arch. für Klin. Med.," vol. lxxvii.

meningitis externa and secondarily inducing a root neuritis and ganglionitis proportionate to the clinical manifestations of the disease.

**Symptoms.**—The clinical manifestations mainly fall within the domain of the motor neurons. The first thing to attract attention is the development of spasmodic stiffness, usually first appearing in the fingers and wrists. After the recurrence of several attacks adults sometimes recognize in malaise, headache, depression, and general pains, premonitions of the on-coming rigidity. The attacks are initiated by a feeling of



Fig. 377.—Infant with mild attack of tetany, showing characteristic spastic position of hands and feet.

prickling, numbness, and some local pain. The spasm comes on slowly and increases gradually in intensity, accompanied by growing discomfort and pain in the muscles. There are no mental features attributable to tetany. The spasms begin peripherally in the fingers and toes, and advance toward the trunk. Ordinarily, they are limited to the limbs, and mainly affect the parts below the elbows and knees. The upper extremities may alone be invaded. In other cases the tonic spasm reaches the roots of the limbs and invades the trunk, and may involve all the body-muscles in very severe cases. Retraction of the head and stibismus are seldom encountered. The contraction is tonic in character and usually persistent during the attack, but may intermit. The attack may last from a few minutes to many hours and occur

several or many times daily. Attacks may cease for intervals of days, weeks, or months, and then reappear, but if latent they can be provoked in the intervals by appropriate manipulation.

The positions and attitudes caused by the spasms of tetany are strikingly peculiar. Ordinarily, the hands are rolled into the cone-shape of the *accoucheur*, the digits flexed at the metacarpal joints and rigidly extended at the interoscal articulations, the thenar and hypothenar eminences approximated. The *wrist* may also be flexed and the hand drawn to the ulnar side. Less commonly the fingers and wrists are extended, or the hand may be made into a fist grasping the thumb, or the thumb may protrude between the index and middle digits. In cases of severity the *elbows* are flexed and adducted strongly against the body. The *feet*, when affected, present a forced equinovarus position, with flexed and sometimes overlapping toes. In extreme cases there is flexion at the *hips and hips*. The *muscles* of the forearms, legs, hands, and feet are tense, firm, and often sensitive. Voluntary movements in the parts are impossible, and passive motion causes pain. When the spasm invades the trunk, intercostal, abdominal, and spinal rigidity may appear and breathing be impeded. In rickety children *turgescens stridens* is



not uncommon; in adults laryngeal spasm constitutes a serious complication. In rare instances spasm in the neck-muscles draws down the chin and the angles of the mouth, and has even produced fatal compression of the air-passages. Solovieff<sup>1</sup> has noticed in adults rhythmic contractions of the *diaphragma* synchronous with the heart-beat and attended by a whistling sound in the left lung. Thoracic and diaphragmatic rigidity may induce asphyxia and even death. Ordinarily, the face escapes. The *sphincters* may be tonically contracted, inducing distipation and anastrea, according to Oble and Sarles,<sup>2</sup> who also noted indican in the urine, due to intestinal fermentation, and confirmed Weiss' observations of the hyperoxiety of the urine. Retention of urine may be the most prominent symptom.<sup>3</sup>

The electrical and mechanical irritability of the motor nerves is peculiarly increased. If pressure be made over the median or ulnar nerves, the spasm in the hand is increased, or during its absence is provoked. Pressure over the brachial artery may have the same effect. This is known as *Trousseau's sign*, and is practically pathognomonic. Gentle tapping on the nerves, as with a percussion hammer, has the same effect. Chvostek discovered that the facial nerve could be aroused in the same manner, causing a facial contortion exactly limited to the distribution of a branch or of the entire nerve, depending upon the location of the blow—*Chvostek's sign*. Erb first described a peculiar exaltation of the electrical excitability, especially to the galvanic current—*Erb's phenomenon*. A single cell, giving but one or two milliamperes of current, may be sufficient to provoke sharp contractions, and a single opening tetanus (A. O. Tc.) is found in this disease alone. Sarbo has, in a single case, noted the myotonic reaction in the triceps, a response that was considered confined to Thomsen's disease. It consists of persistent contraction, lasting some moments after cessation of the galvanic current. The fanning responses are also accentuated in most cases, but many remain about normal. Pressure upon the nerves is more than ordinarily painful, and readily induces persistent paresthesia in their cutaneous distribution. The tapping that causes spasm also induces pain. Schlesinger<sup>4</sup> has directed attention to a *leg sign* which he ranks with the Trousseau phenomenon. If the extended lower extremity during the intervals between spasms is strongly abducted at the hip, in a few moments a painful cramp develops in the knee and a tonic cramp appears in the foot and toes. *Edeema* of the hands and feet and localized perspiration may be encountered. The temperature may be normal, but is often elevated, as might be expected, in the gastro-intestinal cases, and abnormal in the athyroidal state of operative myxedema. Auditory and optic symptoms and trophic changes in the muscles are found only as accidents, but all adult cases are likely to present cataract, and in those dating back to infancy, as is generally the case, dental deformities and cataract are almost invariably observed. Objectively, cutaneous sensibility is normal, and the reflexes are unchanged except when inhibited by the spasmodic state of the muscles.

<sup>1</sup> "Ruski Vrach," 1902.

<sup>2</sup> "La Méd. Inf.," Sept. 15, 1894.

<sup>3</sup> Barkworth, "Jahrb. f. Kinderh.," 1899.

<sup>4</sup> "Wiener klin. Woch.," 1910, No. 9.

**Course.**—The great majority of cases run a mild course to recovery in a few weeks, especially if the underlying cause can be removed, but when tetany develops on gastric dilatation or chronic catarrhal enteritis it is likely to have a protracted course. Appearing generally when the organism is already depressed by malnutrition, it may be a formidable complication, and in the severe cases, where the spasms invade the trunk and implicate the respiratory apparatus, death by asphyxia may result. In pregnancy it is likely to be mild and usually terminates promptly after delivery, but parturition may cause very severe exacerbations of the attack. The lactational cases are usually manageable if the child be weaned and the nutrition of the patient re-established. Tetany in the athyroidal state is frequently fatal; it may be permanent and exceptionally it yields to thyroid and parathyroid feeding. Only when Trousseau's and Chvostek's signs fail can the disease be considered at an end. In fatal cases the spasms increase in frequency, distribution, and intensity, and asphyxia destroys the patient, but death in tetany is more commonly the result of the underlying disease.

**Diagnosis.**—The diagnosis is easy if the characteristic spasms are accompanied by the mechanical and electrical overexcitability of motor and sensory nerves. We have to exclude tetanus, in which the spasms are preceded by stiffness in the masseters which does not subside in the intervals between the spasms. It is also marked by nuchal rigidity and great general irritability. In tetanus the spasm is propagated toward the extremities, usually sparing the hands; in tetany it is centripetal and usually begins in the hands. The phenomena of Trousseau, Chvostek, and Erb are absent in tetanus. *Hysteria* also lacks these special signs and has its own stigmata. *Meningitis* may be mistaken in infants, but presents none of the special signs of tetany.

**Prognosis.**—The prognosis is usually good, but hangs upon the nature and manageability of the underlying cause. It is unfavorable in proportion to the extent and degree of malnutrition present. It is grave in the athyroidal cases, which present a mortality of about 80 per cent. A tendency to recurrence upon renewal of the predisposing state is marked. Thus, some women present tetany in several successive pregnancies, and exacerbations of the predisposing gastro-intestinal state may incite a return of the tetany. Extreme severity of spasms, involvement of the respiratory apparatus, intense manifestation during labor, and cerebral symptoms, as in uremic cases, are of serious import.

**Treatment** must be directed to the removal of the underlying causal state, but it is rarely necessary to interrupt pregnancy. Rickets, gastric dilatation, intestinal catarrh, intestinal parasites, and lactation have their several indications. Hygienic and sanitary conditions must be attentively studied. The spasms are controlled by quiet, rest, and warm baths. Bromids and chloral are useful in the interval, but morphia, and even chloroform, may be required for the attack. When this coincides with labor, the birth must be hastened. Chloroform must be used with circumspection, as it may provoke laryngeal spasm, as may also the employment of the stomach-tube in gastric cases. In the athyroidal cases thyroid feeding sometimes gives relief, but can not be relied upon.



The administration of thyroids in other cases may also be tried. Massage, electricity, and passive movements must be avoided, as they usually intensify the spasms when present, and provoke them if absent. A hot sponge over the larynx may relieve laryngeal spasm, but tracheotomy may be required in rare instances. Odo's<sup>1</sup> attaches much value to the continuous use of calomel, which corrects the gastro-intestinal fermentation and expels toxic substances. Dietetic regulations are often of the first importance. Lactate or acetate of calcium and large quantities of milk should be ordered. Glandular feeding with parathyroids, adrenals, testes, and thyroids have been variously tried without any convincing results. The condition of the bladder should be watched and retention of urine prevented.

## CHAPTER II.

### VASCULAR NEUROSES.

#### RAYNAUD'S DISEASE.

Raynaud, in 1862, described a peculiar dry gangrene, especially in the extremities, which he attributed to a disturbance of the vasomotor apparatus. From present information the condition may better be considered as a generalized one, but with local exaggeration. Naturally, the circulatory disturbance is most apparent in the extremities, as in the fingers, toes, nose, and ears. The arterioles and venules are spastically contracted during the attack. Raynaud described three stages, which he strikingly named local syncope, local asphyxia, and local death. The condition is usually symmetrical, may recede at any stage short of gangrene, and usually appears many times in succession in mild degree before inducing amputation.

**Etiology.**—Females furnish twice as many cases as males and no age is exempt, though most cases occur between twenty and forty-five. Senneker<sup>2</sup> however, after collecting statistics believes that it is as frequent in children as in adults. All varieties of menia are strongly predisposing factors, and a neuropathic makeup is almost invariably present. Other nervous diseases are commonly associated, such as hysteria, epilepsy, tabes, neurasthenia, myelitis, and insanity, especially acute mania. Urticaria, telangiectasis, angioneurotic edema, and scleroderma may be combined in the history or present in the patient. Urticaria and local asphyxia may alternate in the same patient. Heredity is apparent in ten per cent. of the cases showing the same disorder in blood relatives. Nekam<sup>3</sup> reports seven children of one mother, who had been twice married, affected with this vascular defect. Any occupation attended by exposure to cold and wet may play a part in the causation. Any sudden demand

<sup>1</sup> *Lancet*, 1890.

<sup>2</sup> *Paris Thèse*, 1905.

<sup>3</sup> *Neurolog. Centralbl.*, Oct. 1904.

upon the physical strength or powers of resistance may induce the attack. Exposure to cold is the most common immediate cause, but fright, grief, fatigue, typhus, influenza, malaria, and acute infections may induce it. Some consider the ultimate cause of the angiospasm to be an auto-intoxication. It is often associated with scleroderma.

**Symptoms.**—The local symptoms first attract attention. The fingers and toes, less frequently the ears, nose, and lips, or a single finger, appear pale, waxy, bloodless, and glossy. There is usually a feeling of tingling, numbness, and loss of sensibility, as in a finger compressed by an elastic bandage. A needle-prick draws no blood and the finger looks dead; hence the term *digitus mortuus*. After lasting a few minutes to several hours, this condition, often attended by chilliness, nausea, and general discomfort, may pass off or develop the second stage of local asphyxia. The affected part becomes cyanotic, blue-black, and the anemic pressure-trace disappears very slowly. Less commonly the fingers in this stage may puff up, present a vivid red color, be extremely hot, and covered with perspiration. There is now usually more or less neuralgic pain proportional to the cyanosis. Both hands may be simultaneously affected or one may precede the other. One finger may, in the second stage, present the extreme blue-cold cyanosis, its neighbor the turgid, hot condition or the white, syncopic stage. If the second stage persists long enough, several hours at least, small blebs appear, raising the epidermis especially from the pads at the finger-tips, ulceration follows, and dry gangrene may ultimately and destroy the terminal phalanges or entire fingers. Small necrotic patches may form, and, healing slowly, leave cicatrices to mark the attack. The amount of mutilation, fortunately, is often insignificant in relation to the extent of cyanotic tissue. A hand or foot that appears doomed may only lose a few phalanges. The disease may appear in other portions of the body, as in patches over the deltoids, inner aspect of the calves, the heels, malleoli, nates, cheeks, and on the abdomen, rarely, however, going on to gangrene. The genitals and tongue are exceptionally attacked.

Constitutional symptoms rarely default. Intermittent hemoglobinuria, uremia<sup>1</sup> with diminished urea and a lessened quantity of urine, and uremic convulsions are frequently noted. Attacks of colic have been met with during the angiospastic attack. There is no fever at any period except from coincident febrile disease. Cerebral disturbance is very common.<sup>2</sup> Irritability, depression, apathy, incoherence, coma, convulsions, and mania have all been noted. The kidney symptoms and the brain disturbance are apparently due to the angiospasm, which further shows in the narrowed retinal artery, dimness of vision, occasionally occurring hebeticity, tremors, agnesia, and iridoplegia.

**Course and Prognosis.**—The attacks are of variable duration and intensity. A few hours is usually sufficient for the local syncope and asphyxia to develop and subside, but ordinarily another exposure to cold or the incidence of any mental strain or physical fatigue occasions

<sup>1</sup>Arthur: "Lancet," Sept. 26, 1896.

<sup>2</sup>Osley: "Amer. Jour. Med. Sciences," Nov., 1896.



a return. When gangrenous changes have developed, several months are usually consumed in exfoliation and cicatrization, as the process is extremely slow and healing very tardy. In infants the disease is of rapid course and may terminate in death in a few days. In older patients recovery is the rule, but anemia may cause death.

**Diagnosis.**—The diagnosis in a typical case is not difficult. The causes of ordinary gangrene are lacking, but nephritis may be present in Raynaud's disease. Repeated symmetrical local syncope, followed by regional asphyxia and passing away in a few hours, can scarcely be mistaken. Several bouts of lesser degree usually precede the gangrenous variety, often appearing during the winter for years in succession. Hæmoglobinuria or retention of urea greatly assists the diagnosis.

**Treatment.**—The principal indication is to improve the nutrition and reëstablish the general health. Locally, during the attack, the use of warm applications is indicated. The parts may be wrapped in cotton and the temperature properly maintained by artificial heat. Trinitrin and amyl nitrite sometimes give prompt relief. Morphine, hypodermatically, is sometimes required to relieve pain. The local use of the constant electric current has been much advocated, but the warm salt solutions, in which the extremities are directed to be immersed, probably are as active as the electricity. Cushing<sup>1</sup> strongly recommends the application of an elastic or Esmarch bandage to relieve the attack. When the bandage is removed, the parts rapidly fill with blood and the vascular spasm subsides. The treatment of the gangrene is surgical, but it is especially needful to wait for the demarcation line, as it often includes comparatively little of the threatened extremities.

<sup>1</sup> Jour. Nerv. and Ment. Dis., Nov., 1902.

## ACROPARESTHESIA.

Schultze first, in 1890, used the term *acroparesthesia* to describe a condition which had previously been fully recognized by Nothnagel, Bernhardt, Paterson, and others. It occurs generally after middle life in hard-working women who leave the hands constantly wet, as in washing and scrubbing. The paresthetic feeling is usually a tingling, crawling, or burning sensation, sometimes attended by decided pain and most marked night and morning and in winter. The hands are usually alone affected, but the feet may participate. The fingers may be rendered clumsy and awkward. The color of the skin is usually normal, but may be pale, whitened, or reddened. Sensibility is commonly objectively normal, but hyperesthesia and hypesthesia have been observed. Arteries, veins, and nerves are normal. There is no atrophy of muscles or integument, but occasionally a complaint of weakness. The disorder is not serious, but tends to chronicity and may last many years. Cassirer<sup>1</sup> notes only 12 men in 162 cases; and 106 out of 129 cases occurred between the ages of thirty and sixty. All occupations attended by exposure to cold and dampness are represented. The climacteric, alcoholism, influenza, parturition, and uterine extirpation are also noted as causal. The paresthesia is never limited to the distribution of a given nerve, but usually affects all the digits about equally, may extend to the elbows or even to the shoulders, and is bilateral as a general rule. The feet are also affected, but less frequently and less severely.

The **diagnosis** must exclude Raynaud's disease, multiple neuritis, and changes due to severe anemias and syphilis, and scleroderma.

The **prognosis** is not good as to early recovery, but favorable as to any serious resulting condition.

In **treatment** the faradic brush, the static spark, and local galvanism have been accredited with favorable action. General measures, tonics, hydrotherapy, and, most of all, avoidance of occupational causes are mainly to be relied upon. Collins found intestinal disturbances of some sort in 57 out of 100 cases.<sup>2</sup> All such conditions require unflinching attention.

## INTERMITTENT LIMPING.

A condition first described by the French under the term *claudication intermittente*, has been designated *intermittent limping* by the English, *intermittente Hinken* by the Germans, *angiosclerotic paroxysmal myofascitis* by Hignet,<sup>3</sup> and *angust cruris* by Wallon.<sup>4</sup> The condition is manifested in pains and paresthesia, generally in the feet and legs, intensified on attempts at walking, and in severe cases preventing the patient advancing more than a few steps at a time. Usually the feet are more comfortable in a dependent position, and in some cases the patient can only obtain sleep by allowing the feet to hang over the edge of the bed or by sleeping in a sitting posture. A recurring

<sup>1</sup> "Die Vasomotorisch-trophischen Neurosen," Berlin, 1901.

<sup>2</sup> "Med. Rec.," May 31, 1892.

<sup>3</sup> "Deut. Zeit. f. Nervenzellk.," July, 1901.

<sup>4</sup> "Boston Med. and Surg. Jour.," April 3, 1902.



lameness has long been known to veterinary medicine and is found to be due to disease of the aorta, generally an aneurism, interfering with circulation in the hind extremities. In the human race it is always associated with, or perhaps one should say is the manifestation of inefficient circulation in the legs and feet. In the majority of instances no pulse can be obtained in the dorsal artery of the foot, and in a large proportion of the cases the posterior tibial is also pulseless. The pain, which is the chief complaint, is manifested in three different conditions: (1) Pain upon walking; (2) permanent pain, frequently in the form of painful paresthesia while the patient is at rest; (3) the pain which initiates true gangrene.

Göbblam<sup>1</sup> in twenty-four cases found the disorder at twenty-five years of age in the youngest, but usually the condition develops in middle life and rarely after fifty. This author believes that the pathological condition is usually an endarteritis, although it is but seldom that any general arterial sclerosis is present. Erb has insisted upon an inherited disposition, and others contend that there is a congenitally defective arterial system. The abuse of tobacco or alcohol has been rather frequently noted in these cases, but the withdrawal of either or both toxic factors does not appear to better the condition. Syphilis was only found once in the twenty-four cases of Göbblam. The condition is usually symmetrical, and in a few instances the nerves of the part have been found histologically changed slightly, presenting those variations which are encountered in an arteriosclerosis. J. R. Hunt<sup>2</sup> insists that there must be a combination of atherosclerosis and vasomotor instability with a tendency to vascular spasm to enable the full development of the intermittent syndrome characterized by the occurrence of both sensory and motor manifestations during functional activity and rapid relief during rest. Hunt has also described an intermittent lumbar lameness of a similar order due, at least in one instance, to an aortic aneurism just above the iliac bifurcation. Massaut<sup>3</sup> has reported a case in which the upper extremities were similarly affected in a woman of twenty-four and no arterial pulse could be found in either arm. The painful paralysis of the upper extremity recurred upon any continuous use of the parts. In the majority of instances the feet are cold or blanched, but in some cases the parts are intensely reddened, suggesting an erythromelalgia, and in others the alternation between blanching and redness strongly simulates Raynaud's disease. It is not unlikely that the three conditions may be combined. Flat-foot has been observed in a large proportion of these cases.

Medicinal treatment seems to have little value. Even the alkaline iodide, which have such a well-founded reputation in chronic arterial changes, give little assistance. Warm baths, massage, electricity in the form of the galvanic current, withdrawal of tobacco and alcohol, rest and general physical improvement are the measures to be adopted. Gangrene, when once it appears, has a tendency to extend, and early amputation is generally recommended.

<sup>1</sup>"*Neuröl. Centrall.*," March 1, 1901.

<sup>2</sup>"*Med. Rec.*," May 27, 1905.

<sup>3</sup>"*Ann. de la Soc. méd.-chât. d'Arras*," March, April, 1901.

## ANGONEUROTIC EDEMA.

*Angoneurotic edema*, otherwise known as *oedema circumscriptum edemat.*, *oedema non-inflammation edemat.*, *giant urticaria*, *periodic swelling*, *Quincke's disease*, etc., was first definitely described by Milton, in 1876, as *giant urticaria*. Dinkelschker and Quincke, in 1882, gave prominence to it under the above title. In 1892 Collins was able to assemble seventy-five cases and made a critical digest of the literature. Since that time numerous instances have been reported all over the world. It is marked by acute circumscribed swellings of the subcutaneous or submucous tissues, often presents a direct heredity, usually appears in those of a neuropathic diathesis, is often periodic or recurrent, and is commonly attended by gastro-intestinal colics.



Fig. 28.—Angoneurotic edema involves but the lower portion of the face in a very acute individual.

**Etiology.**—The most active predispositions are heredity and exhausting conditions. The neuropathic taint may show itself in antecedent or associated neuroses and psychoses in the family or in the patient, or by the stigmata of degeneracy. This, however, is far from being an absolute rule, and numerous cases give no clue whatever to the origin of the neurotic disturbance. Direct heredity is often marked, as in the series reported by Osler,<sup>1</sup> in which the disease numbered 20 cases, extending through five generations, the series of Milroy,<sup>2</sup> showing six generations and 22 cases out of 97 individuals, the five generations reported by the Crowders,<sup>3</sup> with 28 cases. Continued mental or physical exhaustion often prepares the field for the development of the vascular neurosis. The greatest number of cases appear early in adult life, from twenty to thirty-five years of age; but it has been noted in infancy, or in the marked hereditary cases it may be congenital, as in 20 out of 22 cases of Milroy. Both sexes are affected, but females somewhat more commonly than males. In several cases a decided gouty tendency was present. The urine showed a diminished quantity of urea, and the attacks were beneficially modified by reduction of nitrogenous diet.

As *causing causes*, exposure to cold, intestinal disorders, puberty, the climacteric, masturbation, traumatism, fright, grief, and the action of toxic agents, such as tobacco, alcohol, and malaria, have been noted with considerable frequency. In some instances the malarial intoxication has caused quotidian or tertian attacks. In some they have recurred

<sup>1</sup> *Amer. Jour. Med. Sciences*,<sup>1</sup> April, 1888.

<sup>2</sup> *New York Med. Jour.*,<sup>2</sup> Nov. 5, 1892.

<sup>3</sup> *Arch. Intern. Med.*,<sup>3</sup> December, 1917, p. 840.



every seventh or twelfth day. Aside from such periodicity they have a tendency to develop during the latter half of the night, when the circulatory activity is at its minimum. They also show a seasonal increase in winter and summer, apparently the result of temperature changes and chilling of the skin, and sometimes they follow slight blows. The suggestion has been made by Crowder that the condition is an anaphylactic manifestation in individuals presenting a special protein sensitization.

**Symptoms.**—The swellings of angioneurotic edema generally appear without warning, and reach their maximum in a few minutes or in an hour or two. The most usual locations are the face, lips, tongue, pharynx, genitals, and extremities. The hands are frequently affected. The buttocks, shins, or abdomen may be selected. The swelling is tense, sharply defined, not tender, does not pit on pressure, is whitish or pinkish, and rarely marked by purpuric discolorations. It lasts from a few hours to several days, and vanishes as rapidly as it appears, leaving no trace, or as it leaves one locality a similar swelling may show itself elsewhere without symmetrical or anatomical relation. Several swellings may appear at once. In amount, the swelling varies greatly, but may attain large proportions; hence the name giant swellings. Sometimes nodular swellings as large as hen's eggs are encountered. There is usually a subjective feeling of tension and stiffness in the parts, and some burning, prickling, or itching. If the skin is scratched, urticarial stripes and wheals usually appear, or they may attend the attack or alternate with it.

The tongue, larynx, pharynx, stomach, and intestines are sometimes affected by the swelling, and local discomfort and even serious danger to life may result. Dyspnea, difficulty of swallowing, intestinal and gastric colic are thus induced, and several cases have perished from the laryngeal occlusion. Gastro-intestinal symptoms are very common and appear in from one-half to one-third of the cases. A feeling of uneasiness in the epigastrium is followed by distention, nausea, and obstipation. Colic, cramps, profuse vomiting, and intense thirst ensue. The attack terminates with a colliquative diarrhea. There is often a great increase in the urinary excretion during the attack, and albuminuria and hemoglobinuria are not infrequently encountered. An effusion into the joints sometimes take place. Cerebral symptoms sometimes indicate an edematous involvement of the brain. Lassitude, somnolence, headache, coma, slow pulse, general or localized convulsions, have been noted. The cases of regularly intermittent joint effusion in which a painless swelling of the joint recurs every week or two are of this nature.<sup>1</sup> The attacks may come on at various intervals of days, weeks, or months, or with regularity, and ordinarily between the attacks the previous general condition of health is regained. A blow is sufficient to induce and locate a swelling in some cases, and there is a tendency for the swelling to recur constantly in the same locations. In some of the congenital cases the swelling is practically permanent, general as well as intestinal symptoms being absent. Occasionally there is an association of erythema and angioneurotic edema, the redness and swelling being of similar or dissimilar distribution.

<sup>1</sup> Schlessinger, "Die intermittirenden Gelenkschwellungen," "Nothmann's Spec. Pathol.," 1903.

**Diagnosis.**—The diagnosis can present little difficulty if one is on guard. We must differentiate the erythematous nodosities of rheumatism, the persistent blue and white edemata of hysteria, and the edemata of renal, inflammatory and cardiac origin.

**Prognosis.**—There is little danger unless the larynx be affected, and there is a general tendency for the attacks to cease in advanced years, though sometimes they last for life, or may reappear after a long interval. If traceable to inciting causes, the immediate prognosis is improved. If dependent upon toxic conditions due to alcohol, tobacco, malaria, lithemia, or gastro-intestinal infection, the proper intervention promises good results.

**Treatment.**—If the cause can be discovered and removed, the neurosis promptly yields. Otherwise reliance must be placed on those general measures which are fitted to combat the neuropathic constitution and build up the usually depraved systemic condition. The treatment of the attack is symptomatic. Morphia for gastro-intestinal crises, heat and compression to the swelling, and tracheotomy or intubation if respiratory failure is seriously threatened. Bloodgood<sup>1</sup> reports several cases of persisting angioneurotic erythema cured by deep incisions.

<sup>1</sup> "Johns Hopkins Hospital Bull.," May, 1903.



### CHAPTER III. HYPERTROPHIC NEUROSES.

#### HYPEROSTOSIS CRANII

Virchow used the term *leontiasis ossea*, Starr, *xyglocephalic*, and Putnam, *hyperostosis cranii* to designate certain rare cases in which the bones of the cranium, and less markedly those of the face, undergo enormous enlargement. The process is apparently inflammatory in character, but its symmetrical distribution, association with developmental periods of life, and progressive nature point to a lack of trophic control. Putnam,<sup>1</sup> accepting seven from Baumgarten's list, has been able to tabulate fifteen cases, including that of Edes. Morton Prince<sup>2</sup> believes that this condition is but a part of *ostitis deformans*, with which it is sometimes associated, and that both are of neuropathic



Fig. 228.—(Hyperostosis cranii) (Putnam.)

origin. In the majority the disease appears under thirty years of age, and often in childhood or at puberty. The cranial enlargement is sometimes preceded by inflammations about the head, such as erysipelas, and by trauma. Early symptoms have been headache, drow-

<sup>1</sup> *Amer. Jour. Med. Sciences*,<sup>2</sup> July, 1906.

<sup>2</sup> *Ibid.*, Nov., 1907.

sinus, epileptic attacks, deafness, and blindness. Mental irritability or encephalment is common. Exophthalmos, loss of hearing, facial palsy, optic neuritis, and blindness are due to local or intracranial pressure. The anterior portion of the head is usually most enlarged, and the upper facial bones are more affected than the inferior maxilla. Prognathism is not commonly observed. There is usually a large increase in the bitemporal diameter. In some instances the bones are rather evenly enlarged; in others there are numerous exostotic thickenings, both on the outer and inner surface of the cranium, or on either aspect alone. The cervical vertebrae are sometimes also enlarged. The disease is progressive and not amenable to treatment. Putnam, Starr, and others have tried trephining. Encephalic pressure may in suitable cases be relieved by trephining. Probably a number of different conditions have been grouped under this title. Some instances have shown unmistakable primary disease. The bony enlargement of the head has been associated with persistent infantile conditions, and in later cases with sexual devolution. They may constitute aberrant links between more distinct clinical types of excessive and defective primary activity.

#### HYPERTROPHIC OSTEOARTHROPATHY.

In 1890 Marie and, independently, Bamberger described cases of chronic inflammatory disease attended by skeletal deformities. For such, Marie proposed the term *oséo-arthropathie hypertrophique progressive*. Masanlungo, Thayer, and others insist that the pulmonary factor is by no means essential, and Thayer<sup>1</sup> found it absent in 12 out of 55 typical cases. Other chronic conditions to which it is secondary are syphilis, heart disease, diarrhea, and spinal curies. It has been associated with pthiasis, empyema, bronchiectasis, pulmonary syphilis and acroplasma, pleurisy, bronchitis, and gastric dilatation. The disease presents a multiple osteitis, affecting mainly the terminal phalanges of the hands and feet, the extremities of the long bones of the limbs, and the neighboring joints. The affected bones are enlarged, rarefied, and show the evidence of inflammation. The cartilages are eroded, the synovial membranes thickened. The bones, however, may escape,<sup>2</sup> the enlargement of the finger-ends being due to an excess of subcutaneous fat. The fingers, owing to the globular enlargement of the distal phalanges, have been likened to drumsticks. The nails are greatly enlarged and overhang the thickened finger-ends like a parrot's beak. Usually they are thin and smooth. The joints creak and grate on motion, and the ankles and wrists are ordinarily broadened and thickened. Elbows and knees may be similarly affected, and the toes show deformities and disproportions similar to those of the fingers. The invariable presence of chronic toxicemic states indicates a toxic basis for the trophoneurosis; for instance, Preble<sup>3</sup> noted it in a case of gastrerostasis.

The disease usually is of insidious onset and is frequently attended by rheumatoid pains. It may occur at any age.

The diagnosis is commonly easily made, though at first this dystrophic disease was confused with acromegalia. It does not affect the

<sup>1</sup> *Phila. Med. Jour.*, Nov. 5, 1898.

<sup>2</sup> E. F. Bassant, *Tr. Med. Assn.*, June 1, 1902.

<sup>3</sup> *Medicine*, Jan., 1898.



head, face, or soft parts, and does involve the joints. In acromegalia the fingers and toes are uniformly enlarged, and the wrists and ankles are not disproportionately increased in size. The facies of acromegalia is never seen in pulmonary osteo-arthropathy.

The disease runs a protracted course, but may come to a standstill, or it may recede slightly if the thoracic condition is greatly improved. Desmons and Beraud<sup>1</sup> report some improvement following subcutaneous injections of extracts made from sheep's lungs.

#### LOCALIZED HYPERTROPHIES

**Symmetrical Lipomatosis.**—Occurring commonly in adults, and usually on a background of syphilis or alcoholism, are cases manifesting



Fig. 230.—Symmetrical lipoma: firm mass on left mandible on left maxillary articulation on right side of jaw (face).

localized and symmetrical fatty deposits. These may be tender and the seat of spontaneous pain or comparatively insensitive, and the pain may be quite insignificant. Their favorite location is about the neck, the *diffuse Lipome des Biceps* of the Germans; over the body, as in the case of Hugier, which presented twenty symmetrical pairs; in the axilla and groins; or on the extremities, as in the case reported by Mathieu, one pair over the trochanters, one pair on the inner side of the knees. The adenolipomatosis of Launois and Bensussan<sup>2</sup> (Fig. 231) appears to belong to the same group.

The ill-defined fatty masses are commonly spread out at their borders. Surgical removal of the fatty tumors are sometimes indicated.

<sup>1</sup> *Archives gen. de Méd.* 1891.

<sup>2</sup> *Nouv. Ann. de la Sédn.* 1900.

**Unsymmetrical Hypertrophies.**—In rare instances, usually congenital, and frequently of neurotic ancestry, one side of the body or one extremity, or a portion of an extremity, as a hand or one or more digits, may be disproportionately large. The asymmetry usually increases with the child's growth. In still rarer cases it makes its appearance after birth, and may occur at any time up to maturity. The



Fig. 221.—Unsymmetrical adipospondylia (Lewandowski and Engelke).

hypertrophy usually involves the affected portions en masse, so that the increase in length is proportionate to the breadth and thickness. The principal increase is usually in the adipocellular tissue, but the muscles may be hypertrophic and may show correspondingly increased strength. Usually, however, the muscles are defective. The bones are simply enlarged. Sometimes the hypertrophied parts are warmer than their



normal fellows and may show increased perspiration and evidences of hyperemia. Occasionally there are pigmentary markings. Hemihypertrophy of the face may be encountered, and Friedreich reports a case



Fig. 222.—Hemihypertrophy (isolated hypertrophy) of left (fig. 223) finger (Lillies).

presenting hypertrophied left face and arm and right leg. In rare instances such localized hypertrophies are seen in gigantism and acromegalia. Of their nature we know practically nothing. In some



Fig. 223.—Extreme localized hypertrophy of lower extremities and feet from youth of age (Hilge).

instances it has been attempted to check the overgrowth by compressing the arterial supply and by the injection of astringents, but no good seems to have resulted. Enlarged digits may be amputated.

**Chronic Hereditary Tropho-edema.**—Henry Meigs<sup>1</sup> describes a family in which edema affected eight members, both men and women, distributed through four generations. Four of these cases were observed, and present the same singular affection; namely, a chronic white, firm, and painless edema, appearing at the age of puberty, and affecting especially the feet and legs and sometimes the entire lower members, generally on both sides. He also refers to a remarkable family reported by Milroy,<sup>2</sup> in which in six generations there were twenty-two cases. Other similar family groups have been published, and A. Thomas thinks that one-sided hypertrophies of the body belong to the same category.

The condition in all known instances has caused but little inconvenience, has been attended by no suffering, has not shortened life, and has resisted all forms of treatment.

<sup>1</sup> "Nouv. Icon. de la Syphilis," Dec., 1859.

<sup>2</sup> "New York Med. Rec.," 1852.



## CHAPTER IV.

## INFECTION NEUROSES.

Three diseases temporarily grouped among the neuroses, due to the action of infections, are *tetanus*, *hydrophobia*, and *rabies*. In the first two the infection is capable of experimental propagation; in the last the nature is as yet an inferential matter. All present a preponderance of motor symptoms. Tetanus and hydrophobia are properly surgical conditions, and will be very briefly outlined.

## TETANUS.

Tetanus is an acute infectious disease marked by tonic spasms of the voluntary muscles, usually commencing in those of the jaws; hence the names *trismus* and *lockjaw*.

**Etiology.**—The disease is comparatively more common in hot climates and in the colored races than among Caucasians in temperate and cold regions. This may have relation to the better protection by footwear and clothing in the latter conditions. It spares neither age nor sex, and is a common disease among horses. It may occur endemically. It is probably always introduced traumatically, and can usually be traced to inoculation by objects contaminated by the ground-soil, in which the bacillus of tetanus readily lives. Naturally, the hands and feet are the most common locations of such contaminated abrasions or more extensive lesions. The bacillus first discovered by Nicolaier, and cultivated by Kitasato, is an anaerobic, drumstick-shaped, motile microbe. Culture filtrates contain retaining poisons which are active when inoculated, but not when ingested. Experimental evidence indicates that, like strychnin, their action is mainly upon the spinal cord.<sup>1</sup>

**Morbid Anatomy.**—The condition of the wound presents nothing characteristic, and in the brain and spinal cord the congestion, perivascular exudation, small hemorrhages, and pigmentation of cells sometimes encountered are neither constant nor distinctive. They may even be looked upon as the results of the spasmodic conditions that mark the clinical course of the infection. The same is true of the serous ecephaloses, pulmonary congestions, and muscular ruptures.

**Symptoms.**—From two to twenty days or more after inoculation the first symptoms appear. The intensity of the disease and its fatality are usually in direct proportion to its early onset. Stiffness of the neck and jaw-muscles first appears, limiting mastication, the movement of the tongue, and of the head. Kernig's symptom appears early, and persists continuously throughout the clinical period.<sup>2</sup> Malaise, chilly con-

<sup>1</sup> Wassermann and Takaki, "Berlin Klin. Wochenschr.," June 5, 1908.

<sup>2</sup> Babinski, "Berlin klin. Wochenschr.," 1908, No. 26, 32.

sations, or rigors may antedate the muscular stiffness, but usually do not attract much attention. The muscular spasm increases in intensity and invades the face and trunk. From the tonic action of the zygomatic group the angles of the mouth are retracted in a characteristic sardonic grin that uncovers the teeth, and the jaws can be only slightly separated or may be quite immovable. The head is then retracted, and the entire back may be affected, causing rigidity or, in greater degree, opisthotonos. In some cases the trunk is bent laterally or forward. The lower extremities are usually more affected than the upper, and the forearms and hands are last and least involved. The muscular spasm is chiefly tonic, but if the condition becomes well marked, there are sharp, short, convulsive exacerbations that may reach a most frightful intensity. They are then provoked by the slightest irritation, such as a sudden noise, a bright light, a touch, the jarring of the bed, or any motor effort. They may occur a few times a day or, in extreme cases, with great rapidity, at scarcely noticeable intervals. When these develop they are attended by pain proportional to their intensity and duration. They may impede the thoracic respiratory excursions or induce laryngeal spasm and dyspnea or asphyxia. Often the thoracic or laryngeal spasm induces a hoarse noise, which, taken with the distorted face, rigid limbs, retracted head, and opisthotonic position, presents a frightful picture. Profuse perspiration may be occasioned. The temperature may be normal, slightly increased, or hyperpyrexia may appear and ordinarily precedes a fatal termination. Through it all the mind remains unclouded.

**Varieties.**—*Head tetanus* or *cephalic tetanus* follows wounds upon the head, face, or neck; is usually of prompt appearance after the inoculation; is ordinarily marked by trismus, dysphagia, facial palsy, and respiratory difficulty, a rapid course, and a fatal termination. The modification of tetanus in this form appears to be due to early poisoning of the medulla. The facial palsy that frequently and the oculomotor palsies that sometimes occur indicate nuclear disturbance. The difficulty in swallowing gives a rough resemblance to rabies and has led to the term *tetanus hydrophobicus*. *Tetanus umbilicorum* is usually due to infection of the umbilical stump, and is unknown to aseptic midwifery. *Puerperal tetanus* occurs in parturients. The invasion route is usually through the uterus.

**Diagnosis.**—Given a locus of inoculation, the disease can scarcely be mistaken. When a history of trauma is wanting *hydrophobia* may be suspected, but lacks the jaw-spasm and persistent muscular rigidity. *Strychnia poisoning* is a closer imitator, but has a more rapid onset, more violent and extensive convulsions, trismus is absent, and relaxation occurs between the spasms. *Tetany* affects the hands and feet mainly and primarily and shows a number of special reactions, such as increased electrical excitability and Trousseau's sign. *Hysteria* may imitate tetanus, but ordinarily gives a hysterical history and presents the stigmata of the neurosis. It also usually appears suddenly after a hysterical convulsion, suddenly disappears and recurs, and lacks the nuchal rigidity and mental clearness of tetanus. Bacteriological examination of pus from wounds may make or confirm the diagnosis.

**Prognosis** is always grave and the mortality is over eighty per cent.



Cases appearing before the sixth day usually die; those appearing after the twelfth day are likely to recover. Death results from apnea and heart-strain. Favorable indications are: late onset, limited muscular spasm, absence of respiratory and medullary symptoms, infrequency of convulsions, normal temperature, and ability to receive and assimilate nourishment.

**Treatment.**—If the wound of entry for the tetanus infection is in an unhealthy state, surgical measures of local disinfection are always in order, and usually consist of scraping, cauterization, and the employment of active germicides. Lambert<sup>1</sup> believes that hydrochloric and carbolic acid together furnish the best local application. It often happens that the infection atrium is completely healed, and even early cauterization seems to be of doubtful assistance in checking the disease. The *general management* is of prime importance. The patient should be secluded in a darkened room and every possible excitation be prevented. Alimentation should be carefully maintained by easily digested fluid foods, and, if necessary, by the use of the nasal tube or by rectal injection. Sedative drugs and antispasmodics are indicated, and various ones have cures credited to them. Chloroform and nitrite of amyl are useful to meet the convulsions. Chloral, bromids, morphia, calabar bean, and curare are advised, but must be used with a free hand or omitted entirely. Hot baths sometimes act most soothingly. Active artificial respiration is required in case of dyspnea and threatened asphyxia. *Immunisation* of late years has been attempted by the use of the antitoxins introduced by Tizzoni and Cattani. They have been found practical and reliable. They are valuable in proportion to their early use. Systematic employment of tetanus antitoxin in warfare and civil practice has proved itself of the very greatest service. In the treatment of clinically developed tetanus, antitoxin has been injected into the substance of the brain through trephine openings in order to bring it quickly into operation, but the results hardly warrant the method. Intraspinal, subdural, intravenous, and subcutaneous injections are less objectionable and probably equally efficacious. The removal of spinal fluid and the intradural injection of curare and morphia has been of apparent benefit. Intraspinal injections of magnesium sulphate, 1 c.c. of a 25 per cent. solution to each 25 pounds of body weight, after the method of Meltzer, is strongly recommended by some. Baccelli employed subcutaneous injections of carbolic acid, using a 2 per cent. solution and beginning with a dose of 0.3 to 0.5 c.c., increasing gradually to 1 c.c. every four to six hours, being guided by the symptoms and the urine. At present medicinal preparations are also imperatively demanded in the treatment of tetanus.

<sup>1</sup>"*Amer. Jour. Med. Sciences*," Aug., 1897.

## HYDROPHOBIA.

Hydrophobia is an acute, infectious disease of omnivorous animals, transmissible to man and to other animals by inoculation. It is also known as *rabies* and *lyssa*. The inoculating animals usually are dogs or wolves, but the cat, skunk, and even poultry may carry the disease. The exact nature of the poison is unknown. It undoubtedly is a living contagium. The disease is rare in this country, but seems to be growing more common in the Eastern States, and almost invariably, in man, is the result of bites by rabid dogs. In North Germany, where the muzzling of dogs is rigidly enforced, the disease is almost unknown. It is, therefore, in civilized countries a preventable disease.

**Morbid Anatomy.**—The nervous system frequently shows lesions, but these may be completely lacking and to some extent, when present, are secondary to the disease, following the spasms, dyspnea, and cardiac failure. They consist essentially of vascular disturbances: dilatation, perivascular leukocytic infiltration, ante-mortem intravascular clots, and minute hemorrhages. Such changes are most frequently encountered in the cortex cerebri, the medulla, and cord. They are most intense in the neighborhood of the pneumogastric and hypoglossal nuclei. The perivascular infiltration in this locality may be intense enough to suggest milary abscesses. The presence of Negri's bodies in the nerve-cells of the cord and brain is now looked upon as positive evidence of rabies. Their exact nature is still undetermined. They are round or oval, from 1 to 20 microns in size, and very persistent in character. Negri supposed them to be parasitic. It is probable from various analogies with poliomyelitis that similar almost ultramicroscopical organisms will eventually be isolated in this terrible disease. The salivary glands and kidneys frequently show round-celled infiltration and the mucous membrane of the pharynx and larynx is commonly congested.

**Symptoms.**—*Incubation* requires a variable period of from two weeks to six months, and there are reported cases occurring twelve and even eighteen months after inoculation, the virus having remained dormant. The ordinary incubation period is six weeks to two months. The length of incubation time, according to Hoesley, is modified by a number of factors: (1) It is shorter in children than in adults. (2) Wounds of the face, neck, head, and hands, and the unclashed parts are especially dangerous, and the disease then develops promptly. (3) Punctures are the most dangerous; lacerations are serious in proportion to their extent. (4) The bites of rabid animals are serious in this order: Wolf, cat, dog, and other animals. About fifteen per cent. of the persons bitten by dogs known to be rabid develop hydrophobia.

*Rabies* varies in intensity in both animals and man. In cases of great severity paralytic features develop early, there is little excitement, and death promptly supervenes. When the poisoning is less profound, the disease runs a longer course, and presents a period of great motor and cerebral excitement. In man the incubation of the disease is frequently marked by irritation about the wound, with pain or numbness. Usually there is headache, depression, loss of appetite, irritability, sleeplessness, and anxiety. The pulse and temperature may be slightly increased. Bright lights, noises, and slight excitement of any sort are



shunned owing to the increased sensibility. Stiffness of the throat-muscles and difficulty in swallowing are noticed. A period of excitement then usually develops, when, in rare instances, the central apparatus may be overwhelmed and the paralytic form, with ascending paraplegia and heart-failure, terminates the case within a comparatively few hours. In the excited period there is great motor restlessness and hypersensibility; spasms affecting the throat are induced by any fictitious stimulus, and swallowing becomes impossible, so that fluids are shunned, and the sight of them may even become unbearable; hence the name, *hydrophobia*. Noises, lights, a breath of air, may provoke the spasms, and it may involve the larynx and thorax, producing dyspnoea, cyanosis, and an alarming asphyxia that even tracheotomy may fail to relieve. The respiratory and deglutition spasms are often attended by hoarse sounds and peculiar noises, that have been thought by excited laymen to resemble the barking of dogs. Saliva accumulates in the mouth or drips from its corners, partly from an increased secretion, but mainly from difficulty in swallowing. Hallucinations and delirium are frequently present, but the mind may be quite clear and the patient quiet between the paroxysms. The temperature commonly ascends to  $100^{\circ}$  or  $103^{\circ}$ , but the disease may be afebrile throughout. The spasms gradually become wide-spread and tetanoid, but rarely affecting the jaw and face, and relaxation occurs in the intervals. After one to three days they are followed by the paralytic period, and spasms no longer occur. An ascending paraplegia is commonly presented. Quiet, stupor, and coma follow; the heart's action becomes greatly weakened, and death follows syncope.

**Diagnosis.**—The diagnosis of a well-developed case, with motor excitement and a history of a bite by a rabid animal, can be readily made. We have to exclude *tetanus*, in which there is usually a very recent trauma, inspiratory spasms, constant rigidity, and little or no difficulty in swallowing. *Lupulobdix* is a hysterical condition occurring in a neurotic, and is a variety of hypochondriasis with the fixed idea of rabies. The attacks that occur are hysterical exaggerations of the newspaper accounts of rabid patients, and such cases can usually be deciphered by the concomitant indications of hysteria. Every such case should, however, be carefully watched if it is reasonably certain that the patient has been bitten by an animal suspected of rabies. In every case it is also important to diagnose the condition in the alleged rabid animal, and this can be done with certainty if the Negri bodies are found, or inoculation experiments can be made.

In case of a bite by a dog, the animal should be placed under observation, if possible, and not destroyed. The saliva being highly poisonous, a little of it may be inoculated into the skin of a dog or rabbit, and precise results obtained. In dogs, the paralytic form of rabies is the most common, the furious form exceptional. The early symptoms in the dog consist of dullness, irritability, and restlessness, the bark becomes metallic, food is neglected, but bits of wood, dirt, and other indigestible articles are sometimes swallowed. If furor develops the dog runs, snapping at everything in its way, especially attacking other dogs. Children, being unaware of danger and less able to escape, are more

frequently bitten than adults. After a day or two, or a few hours only in some cases, paralytic symptoms set in. The animal becomes uncertain on its legs, the hinder limbs give way, and the paralysis becomes general, ending in death. The excited stage is commonly transient, and paralytic conditions promptly develop.

**Treatment.**—An inoculated wound, or one reasonably suspected, should be treated locally, at the first possible moment, by thorough cauterization, or excision, if practicable. The use of a ligature above the wound when on an extremity, suction, and free bleeding are also of value. The actual cautery, a bunch of burning matches, strong carbolic or nitric acid, may be used. *Immunization* by Pasteur's method is now practicable in most large cities. The sooner it is undertaken, the more successful it is likely to be. Less than one-half of one per cent. of all cases treated in this way have died of rabies. When hydrophobia develops in man, about the same treatment is required as in tetanus. There is no danger to attendants, and no case is on record where the disease has been transmitted from man to man. Ferrible physical control is practically never needed. If food and drink can be swallowed, it should be freely given, but rectal alimentation is usually required. Chloroform, bromids, morphia, and sedatives are palliative only. The disease is invariably fatal.

#### CHOREA.

It is desired to limit the term "chorea" to a definite morbid entity. The choreiform features of hysteria which mark epidemics of St. Vitus' dance, hereditary chorea, or the so-called chorea major of Huntington, the electric chorea of Dublin, habit chorea or the malalie des fies, and the various forms of myoclonus should be carefully distinguished from minor chorea or the chorea of Sydenham, with which we are now to deal. It is commonly called St. Vitus' dance, but that term may better be reserved for the hysterical forms.

"Chorea," judicially says Osler,<sup>1</sup> "is an acute disease of childhood, rarely of adults and of the aged. Characterized by irregular, involuntary movements, a variable amount of physical disturbance, and associated very often with arthritis and endocarditis. The disease is usually considered as a neurosis, but the clinical characters of the severe cases and the frequent heart and joint complications have suggested to many recent writers that it may be due to a specific poison."

**Etiology.**—*Predisposing Causes.*—*Sex.*—Girls are affected somewhat more than twice as frequently as boys, and the disproportion becomes even greater after puberty. The vast majority of cases develop between five and sixteen years of age. Chorea is comparatively rare before the age of four. The reported congenital cases are usually mistaken instances of organic cerebral disease. After twenty the disease is most rare, but may appear even in advanced years. The great majority of reported senile cases, however, are instances of motor disturbance symptomatic of organic brain lesions. It is somewhat more common in unhygienic and cramped conditions of life, and hence in urban communities, but spares no social grade or locality. Chorea is extremely rare in

<sup>1</sup>On "Chorea," Philadelphia, 1894.



the dark-skinned races on this continent. Negroes and Indians of full blood are very seldom affected. The seasonal relations of the disease are most interesting. According to Lewis, the frequency of chorea reaches its lowest curve in November, but rises rapidly in December, remains stationary in January and February, mounts still higher—to its peak—in March, falls in April, again rises in May, and then gradually declines to its November starting-point. This trace corresponds with that of rheumatism, the general amount of sickness, and climatic vicissitudes. The hereditary make-up plays a distinct predisposing rôle, so that we learn to expect a history of various neuroses and psychoses in the family, and of "nervousness" in the patient. Chorea commonly shows some of the so-called stigmata of degeneracy and often give a history of pavor nocturnus, enuresis, infantile convulsions, febrile deliriums, impressionability, and mental precocity. It is not rare to find that the mother has had chorea.

*Leading Causes.*—*Fright, worry*, especially at school; *mental shock* and *atrois* generally, and *vervately* particularly, are frequently alleged inciting causes, but on close analysis usually retain but little significance. Very frequently the early mental and even motor symptoms of the disease and of rheumatism will be found to have antedated the psychic trauma that precipitated the choreic storm. *Inhibition* plays a most insignificant, if not entirely negative, rôle. A case of true chorea in a school or home for children may, however, start an epidemic of hysterical St. Vitus' dance, or of hysterical rhythmic spasms. Hysteria may, indeed, ape chorea to the minutest detail, and they may be associated. *Tremulation* can not be dissociated from mental shock, and parents always assiduously seek for some such incident and cause. Its real value as an etiological factor is difficult to estimate, but seems slight, and often the particular injury is but one of the insignificant mishaps of every-day life in childhood. *Light irritation*, prominently urged by the older writers, arising from the intestines, and particularly from worms, is seldom found, but should not be overlooked. Diseased conditions in the nasopharynx are more likely to induce ties than chorea, and the same may be said of eye-strain, the fetish of some recent writers. Tonsillar and adenoid disease, however, is on a closely related footing with both chorea and rheumatism. If the ocular apparatus is unbalanced or refractive errors are found, they should be relieved here as well as elsewhere. It is not unreasonable to suppose that their deleterious influence may protract the nervous manifestations of chorea, or may predispose to it by lowering the general tone. *Pregnancy* appears capable of causing a recurrence of chorea or of favoring its development and modifying its course. The chorea of pregnancy furnishes one of the varieties of the disease, and accounts in part for the larger proportion of female cases in adult years. As strongly urged by Tourette, many such cases are purely hysterical, though commonly considered choreic. Chorea is sometimes preceded or accompanied by the *infectious diathesis* of childhood, as might naturally be expected, given the fact that chorea is especially a malady of early life. The relationship is mainly one of coincidence. They may, however, modify the chorea. Occurring during its early period, they tend to intensify it, but if it be on the wane, they

seem not rarely to hasten its regression. The favorable influence, if it exists, may perhaps be attributed to the forced rest they necessitate, and we are to see that rest is the most important element in the treatment of chorea. *Anorexia* and *malnutrition* sometimes precede chorea, and may furnish a certain liability to its invasion, but usually they follow its development and are symptomatic of it.

**Rheumatism** (*Cardiopathy, Arthritis*).—The questions arising regarding the relations existing between rheumatism and chorea are interesting and practically important. In order to start aright, it should be stated that rheumatism is here meant to broadly signify the acute manifestations of an infectious process that is marked by inflammatory disturbances of articular, serous, and endocardial surfaces. This is presumably a heteropathic disorder, but in most instances due to the activity of an unknown specific micro-organism; unless we accept as such the streptococci isolated by Triboulet, Wassermann, Pain and Peyton, and Benton and Walker, or consider it some modification or variant of the ordinary streptococcus, according to Rosenow. It has long been observed that acute rheumatism and cardiac lesions may precede, attend, or follow chorea. Different observers find such relation in widely varying ratio. One sees rheumatism in "growing pains", another requires that all the articular manifestations should be present to justify its recognition. The most reliable statistics give a rheumatic association in about 20 per cent. of all cases. Thus, Osler, 21 per cent. and 18.24 per cent. in two series; Townsend, 21; Crandall, 34; Starr, 18; the British Collective Investigation, 22 per cent. Usually the arthritis precedes the chorea, rarely it follows, and exceptionally it develops during the progress of the motor disturbance. If the milder manifestations of rheumatism are accepted in this connection and allowances made for the unrecognized cases, it is permissible to say that it is associated with chorea in one-half of all cases. Fatal cases of chorea almost invariably show endocardial vegetations which differ in no particular from those due to rheumatic endocarditis, yet such cases give a rheumatic history in only about 25 per cent.

**Pathogenesis.**—It is needless to even enumerate all the theories that have been advanced regarding the nature of chorea. Those now advocated may be divided into the neurotic, the infectious, and the rheumatic theories. Granting that acute rheumatism is an infectious disease, the third division is embraced in the second. The neurotic theory is based largely upon the appearance of the disease during the years of active growth, the common neurotic peculiarities and antecedents of the patients, the incitement by mental shocks, the psychical symptoms, and the complexity of the nervous disorders, which embrace motor, reflex, and sensory troubles. Even the arthritis has been referred to a nervous source. Charcot said it was the old question of arthritis in combination with nervous diseases. Joffroy denominates chorea a cerebri-spinal neurosis of the period of growth.

The theory of infection rests upon the influence of age, sex, and season; the association of chorea with other infections, its intimate relation with rheumatism; the infectious aspects of fatal cases clinically, and the presence of endocarditis at practically every autopsy, often accom-



panied by pericarditis, pleurisy, parotitis, abscess formation, and other septic processes. Finally, there are those who, following Kirkes, Roger, and others, see in chorea only a manifestation of rheumatism. This theory of identity is maintained by Charcot.<sup>1</sup> He says: "The postulated toxin (x) being accepted as an essential element in the causation of rheumatism, depressing conditions (y) determine the first position or focus (z) of the disorder. . . . If (y) is a fright, shock, or intense excitement, (z) will be the nervous system; in the developing brain of a child the result is usually chorea; in adults it may be delirium or coma, perhaps hyperpyrexia. . . . Wringing of the feet always causes arthritis first in the lower extremities; of shoulders, in the upper extremities." The embolic theory of Kirkes, upheld by the experiments of Mowry, was based on the supposition that endocarditis always preceded chorea, which can not be maintained.

As to the precise location of the disease in the nervous system, the mental symptoms, frequent monoplegic or unilateral distribution of the chorea, and the practical exemption of the trophic functions of the lower motor neuron, with absence of sensory disturbance, point to the cerebral cortex. Wood, from experiments and investigations in canine chorea, located the disturbance in the spinal gray, but this disease is not identical with human chorea.

Regarding the nature of the specific microbe in chorea, nothing definite can now be offered. In 1891 Pinesse<sup>2</sup> cultivated a rod-shaped microbe from the cord and brain of a fatal case of chorea, which, injected into animals, produced apathy, then tremor, then convulsive movements, and, finally, death. Autopsical search in these animals detected this bacillus exclusively in the nervous apparatus. This finding has not been confirmed. Duna<sup>3</sup> has found a diplococcus in the meninges of a fatal case, but as subacute leptomeningitis was present, it was possibly or probably the specific micrococcus of that disease. Various streptococci have also been noted. Westphal, Wassermann, and Malkoff<sup>4</sup> isolated from the blood, brain, and cardiac vegetation of a fatal case of chorea a micrococcus which produced arthritis in animals. Rosenow, already mentioned, leans to the opinion that a variation of streptococcus is the essential organism.

**Morbid Anatomy.**—As chorea is seldom fatal, the post-mortem changes found in the extraordinarily severe cases that result in death can hardly be supposed to fairly represent the anatomy of the disease. Even in such cases there are no uniform or characteristic changes. In the brain and spinal cord intense hyperemia, peri-arterial exudations, minute hemorrhages, softened spots, and occasional emboli have been noted, especially in the deeper portions of the motor tracts in and about the basal ganglia. The *chorea corporalis*, first described by Elsässer, and often found by others, are equally developed in various infections, as proved by Manasse, who found them in twenty autopsies upon septic subjects, and was able to produce them in dogs by putrescent intravenous injections. They are hyaline in chemical reaction, develop in and around the arterioles and in the perivascular spaces throughout the brain and cord, and furnish another argument for the infectious char-

<sup>1</sup> "British Med. Jour.," Sept. 18, 1896.

<sup>2</sup> "Ann. Jour. Med. Sci.," Jan., 1891.

<sup>3</sup> "La Riforma Med.," July 14, 1891.

<sup>4</sup> "Berlin. Klin. Woch.," No. 29, 1899.

acter of chorea. Turner<sup>1</sup> describes swellings and opacities in the cortical pyramidal cells. One of his five cases had puerperal sepsis and two albuminuria, both of which might account for the cellular changes. The two remaining cases, if identical, may be taken as showing changes of a septic or infectious nature. The heart is more often diseased in the fatal cases of chorea than in any other malady whatsoever. Osler, in the 73 cases collected by him, finds cardiac lesions recorded in over 80 per cent., consisting of recent endocarditis, 62 cases, with pericarditis 19 times; pericarditis alone in 2 cases; chronic mitral endocarditis twice, and fatty heart once. The ordinary endocardial appearance is a row of papular granulations at the mitral orifice. All varieties of incidental and accidental septic conditions are reported in the literature. Staphylococcus aureus and pyogenes have been noted by Guicetti; staphylococcus aureus and streptococcus by Reichardt. Preopryensky<sup>2</sup> claims to have found streptococci in the blood in two cases, both of which were benefited by the use of antistreptococcus serum. Benton and Walker<sup>3</sup> claim to have found a specific streptococcus in chorea and acute rheumatism reacting in certain respects differently from the streptococcus of human septicæmia.

**Symptoms.**—Chorea is commonly of insidious onset, but its motor symptoms may appear abruptly—that is, within a few hours or days after a fright or other mental disturbance. In the large majority of cases parents can easily recall that for a period of days or weeks before motor disturbance attracted their attention the child had been peevish, obstinate, apprehensive, easily displeased, and less companionable with its playmates. Perverseness and moral oddities sometimes appear. Very commonly the sleep has been disturbed and broken by distressing dreams or actual nocturnal pavor. More often there is more restlessness and difficulty in getting to sleep. This *prodromic period* should be carefully investigated, as it is likely to give valuable warning in case of recurrent attacks. At school the child becomes inattentive and forgetful, and finds increasing difficulty with its studies. If reprimanded, there is undue depression or unusual emotion or capriciousness. The inability to study, due to the lack of mental concentration, leads too often to the supposition of overstudy; but it is particularly among the bright, precocious, and easily stimulated school-children that chorea is likely to appear. These are exactly the children whose mental development should be retarded, and it is among these that forcing methods in school-work are most baneful.

After a widely varying period the motor *choreic features* appear. These at first consist ordinarily of sudden, unwillful, slight movements or unexpected relaxation of muscular contraction. The child appears unbalanced, upset; its cup, drops articles from its hands, and lays itself liable to admonition, and, unfortunately, in some instances, to chastisement, which may bring down the choreic storm in a burst of motor twitchings, grimacings, and spasmodic disturbance. Lacking cause of sudden exacerbation, the movements gradually become more pronounced

<sup>1</sup> Path. Soc. Trans., 1882, vol. xlii.

<sup>2</sup> La Sève Méd., Dec. 10, 1902.

<sup>3</sup> Brit. Med. Jour., Jan. 31, 1903.



and bizarre. There is often complaint of fatigue, pain in the limbs or joints, and frequently loss of appetite and constipation are early noted.

**Motor Features.**—The choreic movements ordinarily begin in the muscles of the hands and forearm, but the face is soon, sometimes first, affected, and then the lower extremities, shoulders, and trunk, in varying order. Occasionally, the chorea is confined to a single extremity or to one side of the body, or, beginning on one side, may invade the other, subsiding or persisting in the original territory. Commonly, both sides are not equally involved. As a rule, the affected muscles show three important functional modifications: (1) Unwilled but conscious twitchings or spasms; (2) inability to maintain steady contraction, and (3) loss of power. The choreic movements vary not only in distribution, but in intensity at different times and in different cases. We may first consider a case of average severity. The spontaneous actions may be described as disordered, irregular, arrhythmic, of considerable amplitude, and of a rapidity between tics and athetosis. They cease during sleep, though there may be great restlessness. They may even prevent sleep. They can be slightly controlled during the execution of voluntary movements, but are excited and exaggerated by embarrassment and any emotional excitement. Mitchell and Rhein<sup>2</sup> define several varieties of choreic cases depending upon motor peculiarities: "(1) Cases which show, at some stage or throughout the attack, an absence of movements during rest. (2) Cases with continuous movements during rest, but increased by intentional effort. (3) Cases with severe choreic movements, entirely disappearing during muscular acts. (4) Cases in which the movements are unaltered by muscular efforts. (5) Cases presenting several of these phases at different times." In the face they usually cause bilateral grimacing, especially affecting the lips and nose, less frequently involving the muscles of the brow and eyelids. The lips may be quickly pursed up or retracted, the tongue protruded and retracted, the teeth snapped together. In this way speech is impaired and becomes halting or explosive, due entirely to faults of articulation, as the larynx is practically never involved. Swallowing is sometimes difficult, mainly, however, on account of the choreic movements of the lips, cheeks, tongue, and palate. The tongue is usually affected very early, and in perhaps the majority of cases the choreic movements persist in this organ after they have elsewhere disappeared. Ordinarily, if the patient is asked to show his tongue the chorea is at once provoked in the facial muscles, but the tongue, too, is animated by incoördinate writhings that appear, subside, reappear, and usually end in its sudden retraction and the quick closing of the mouth. In very exceptional cases the ocular muscles are implicated, causing momentary diplopia, and movements in the iris have been seen attending momentary confusion of vision. Sluggishness of pupillary contraction to light is not uncommon, and a few cases of retinal embolism are recorded.<sup>3</sup>

In the upper extremities the choreic twitching is most and first developed in the fingers, which move individually or together, separate and close, extend or flex, with more or less disorder. Pronation and supina-

<sup>1</sup> "Fla. Med. Jour.," Jan. 22, 1896.

<sup>2</sup> H. Thomas, "Johns Hopkins Hosp. Bull.," (Oct., 1900).

tion are more common than wrist movements, and the shoulders are more affected than the elbows. Indeed, it is rare that shrugging of one or both shoulders does not take place. When the chorea is well marked, objects are grasped with difficulty. The hand approaches them by zig-zags and suddenly swoops down upon them. Finally, prehension may be impossible, and the patients can no longer feed themselves, or spill everything they attempt to carry to the mouth. The *lower extremities* are usually less affected, but, as in the upper members, the digits show the most disturbance, flexing, extending, or separating in disorder. The big toe and the thumb are most active, as a rule. The *station* is cerebral unsteady, sometimes uncertain, and rarely impossible, by the movements of the legs and feet. In walking the gait is often peculiarly disturbed. The steps are unequal in length and irregular in rhythm. The feet may be jerked from the direct line of advance, raised too high, or brought down too vigorously, but never rhythmically. The knees are not always firmly supported, so that altogether there is sometimes presented a peculiar resemblance to the many-jointed action of marionettes danced on a string, which, taken with the grimaces and with the convulsions of the hands, recalls the clownishness Sydenham so graphically described. The muscles of the *trunk and neck* do not escape, and may cause nodding<sup>1</sup> and headings that are often most apparent when the patient is seated. The *diaphragm* and the *thoracic muscles* are commonly involved, causing irregularity of respiration and sometimes spasmodic noises, or there may be peculiar involuntary clucking or swallowing sounds. Graves<sup>2</sup> has made a painstaking study of these respiratory irregularities which, by suitable self-registering apparatus, he finds practically always present, even in the very mild cases, and, what is a matter of more importance, that they persist long after the more noticeable symptoms disappear. In this way many recurrences are shown to be merely relapses of the original attack. The choreic movements may be so continuous and severe as to prevent sleep, to confine the patient to bed, and to cause innumerable bruises and excoriations by friction or by rough contact with hard substances, such as the walls and furniture.

If the patient is directed to grasp the physician's hand and hold firmly, *involuntary pressure* will at once be observed. Relaxation or sudden increase of muscular tension, or both, are noticed. Of still greater importance is a *loss of muscular power*, which is practically always present in muscles affected by chorea, as can be clearly shown in the unilateral cases. This may reach a loss of fifty or seventy per cent., and in the paralytic form of chorea it constitutes the major motor difficulty. The parietic feature of chorea explains the ready fatigue and accentuates the need of rest in the management of the disorder. Russell<sup>3</sup> notes that the *handwriting* may be (1) nearly choreic, (2) may be very good even when choreic disturbance is well marked, (3) may be almost impossible though choreic movements are extremely slight, (4) may be entirely unintelligible though control of the pen is good, and (5) may present pure motor apraxia.

The *sphincters* are never affected except in the last stages of the fatal cases. Objectively, sensibility is normal, as a rule, and any considerable

<sup>1</sup> Jour. A. M. A., Aug. 30, 1900.

<sup>2</sup> Lancet, April 1, 1899.



anesthesia or dysesthesia should cause a suspicion of hysteria. Triloulet laid much importance on sensitive spots beside the spinal processes of the vertebrae and over the intercostal nerves, but they are inconstant. The electric excitability of the nerves and muscles is sometimes increased, and the areolar closing contraction may equal the similar catlethal response. General loss of flesh is common, but localized muscular atrophy is very seldom found. Sherr<sup>1</sup> calls attention to a jerky response upon eliciting the knee phenomenon, otherwise the reflexes are unaffected.

**Mental Disturbances.**—Aside from the temperamental, moral, and affective changes so common in the prodromal stages, and which may persist throughout the attack, other mental disturbances may rarely occur. The former are, in a sense, proper to chorea, and with the myasthenia may cause a marked change in the *facies*, which presents an insane and stupid appearance, often at great variance with the normal attributes of the child. Some of this may be due to the weakness of the facial muscles, but mainly it is consistent with the lethargy, irritability, and weakened mentality in such cases. *Hallucinations* of sight are frequently, and of the other senses rarely, noted. They usually appear toward or during the night. In the grave cases, when the *choreic stasis* is produced, the temperature elevated, and the muscular activity at its height, *delirious*, commonly of a hallucinatory character, is often present. Occasionally, psychoses of various forms are encountered—the concomitant manifestations of degenerative and toxicity.

**Cardiac Disorders.**—In chorea the heart frequently presents clinical symptoms, and apparently is much oftener involved than auscultation indicates. This is shown by the considerable percentage of cardiac lesions in fatal cases without cardiac symptoms, and the astonishing number of cases of chorea which show organic heart-lesions upon examination years after the chorea has disappeared. During chorea heart-murmurs are present in about one-third of the cases, and are functional or organic. In addition, there are disturbances of rhythm, rapid action, and pain. Galbi<sup>2</sup> calls particular attention to the variability of the cardiac diameters during chorea and their wide and rapid changes under insignificant causes.

**Functional murmurs** are most commonly heard at the base and to the left of the sternum, with the systole usually most intense over the pulmonary valves, and often attended by rapid heart-action. Anemic murmurs over the tricuspid propagated into the neck are not infrequent, and are often associated with an increased area of cardiac dullness. They may only be noticeable with the patient in the horizontal position. *Organic murmurs* are usually the result of endocarditis, most often affecting the mitral orifice, systolic in point of time, and with greatest apical intensity. *Palpitation* and cardiac or precordial pain are, on the whole, exceptional. An *abnormal rhythm* is a very common observation in chorea. This has been attributed to chorea affecting the heart-muscle, but is more reasonably referable to functional disturbance and respiratory irregularities, which are very frequent in this disease. Very often the frequency of the heart-beats is the same in both the horizontal and vertical positions. A rapid heart sometimes persists after the attack.

<sup>1</sup>"*Albany Med. Ann.*," May, 1897.

<sup>2</sup>"*B. Policlinico*," Nov. 21, 1901.

Ordinarily, the endocarditis develops during the evolution of the chorea, and is sometimes attended by joint symptoms. Organic heart disease seems to be most frequent from five to ten years of age, but this corresponds to the period of the greatest frequency of chorea. The customary post-mortem findings have been already noted. Of equal or greater importance are the postchoreic observations. Thus, Mackenzie found 95.6 per cent. of thirty-one cases of chorea examined from one to five years after the attack marked by organic cardiac lesions. Osler, in a more extended and very closely scrutinized series, found 51½ per cent. of postchoreic cardiopathies. The same thing is shown by the long-recognized fact of an increasing proportion of cardiac diseases in the subjects of repeated attacks of chorea. The practical deduction points the need of systematically watching the heart throughout the course of, and for a long time subsequent to, the attack of chorea.

The general state in chorea is commonly reduced. Often, but by no means always, there is some degree of physical depression previous to the onset. Anemia, loss of appetite, gastric indigestion, sluggishness of the bowels, and dryness of the skin are usually developed early in the choreic attack, and are sometimes induced or increased by the injudicious use of arsenic. Ordinarily, chorea is afebrile. In the choreic status and in cases of mixed infection or concomitant sepsis, the temperature rises and is proportionate to its cause.

**Course.**—The course of chorea, whether of insidious or of abrupt onset, is usually marked by exacerbations and remissions of the peculiar movements. In the majority of cases, after reaching various grades of severity, the chorea gradually declines, leaving the lower extremities, the upper extremities, the trunk, the face, and the tongue, usually in the order mentioned. At a time when the movements no longer occur spontaneously they may still be provoked in the face, and especially in the tongue, by directing it to be vigorously protruded. The average duration of the disease is from six weeks to four months, but cases lasting two weeks or less and others lasting six to eighteen months are not very rare. The common termination of chorea is in complete recovery, though death occurs in rare instances. A fatal termination results in the very severe cases in which the choreic status exhausts the patient, or more frequently from complications, especially on the part of the heart. Cerebral hemorrhage and softening and concurrent infections may lead to a fatal termination. In other and also very exceptional cases the chorea becomes *chronica*. It may also be followed by a habit spasm or tic.

**Recurrence** in chorea is so common that it is always to be expected, and occurs in over one-third of all cases. Up to the third attack both sexes show their usual relative susceptibility, but beyond that number the preponderance of girls and women rapidly advances in the lists. It is rare to find a male presenting more than 3 attacks, but 10, 12, and more attacks in females are not very rare. Some patients present chorea every spring for several years. As a rule, succeeding attacks grow progressively shorter. She gives the average duration as 138, 80, and 55 days in the first three attacks, respectively. It is to be doubted that some of the alleged repeated attacks are such in fact. In many instances a close examination of the patient in the interval will enable



the observer to evoke choreic traces in the face, tongue, and extremities, though parents, teachers, and patients insist that complete recovery has taken place. In such instances a recurrence, strictly speaking, is an exacerbation. Choreic girls are also later liable to gestational chorea.

**Forms.**—Sydenham's chorea presents several forms or modifications that require individual mention. We may distinguish (1) the common form, which has been the basis of the above description; (2) the grave form, (3) the gestational, and (4) the paralytic forms.

*The grave form, chorea gravis*, is marked by an intensification of all the motor and usually of the mental manifestations of ordinary chorea. It is ordinarily of acute and intense onset, or may occur as a sudden recurrence or intense exacerbation of the common type. The choreic movements are wide-spread, continuous, and violent. They may disturb sleep, or prevent it altogether. The patient usually is unable to stand or to sit on a chair, and may be so violently agitated as to be thrown about and off the bed. Swallowing and speech are interfered with or rendered impossible. Excoriations, bruises, and other injuries result. Infections may thus occur, inducing suppurations and erysipelas. Fever arises; there is delirium, sometimes of maniacal intensity. Such a continued, exalted, intensified, chorea has been denominated the *chorea stans*. The choreic state, thus constituted, persists for days, and finally subsides, death often following coma. It differs only in degree from the milder attacks, and all gradations may be encountered. Mental disturbance at intervals, or more or less continuous, may be encountered, varying between mild momentary delirium and wild mania. Delusions and hallucinations are not uncommon in this phase of mental disorder and may be presented side by side with an appearance of fairly good mental balance and self-control. The maniacal cases usually terminate fatally.

*The chorea of pregnancy, chorea gravidarum*, usually appears in young primiparae, commonly during the first half of pregnancy, and is frequently preceded by a history of chorea in earlier life. The predisposing and exciting causes of the common form also obtain here, and the symptomatology is much the same. Cardiac complications are the rule. The motor agitation is usually intense, and there is commonly involvement of the pharyngeal and respiratory apparatus, less often of the laryngeal mechanism. Abdominal and vaginal palpation usually intensify the choreic trouble, and the fetal movements sometimes have the same result. Mental disturbances and affective changes are nearly always present, and tend to persist after parturition or may only develop post partum. Parturition may diminish, augment, or fail to affect the chorea, but, on the other hand, the chorea may cause premature birth and abortion. Ordinarily, chorea, once established, persists until the nuchal is evacuated and may continue during lactation. It is much more serious than the ordinary form, and terminates fatally in about twenty per cent. of all cases. In some patients it recurs at each subsequent pregnancy.

*Paralytic chorea*, called *hup chorea* by English writers, is probably more frequent than reports would indicate. The parietic element in chorea has been particularly insisted upon. In this form it is the dominant condition, and the choreic movements are insignificant. It has a predilection for young children, and is most frequent at about six or

seven years of age. Commonly, some infectious malady seems to act as a provoking cause or complication. The premonitory stage corresponds to that of the ordinary type. Ordinarily, the paresis appears early and may or may not be preceded by choreic twitching. A monoplegic, hemiplegic, or paraplegic distribution may be presented, but monoparesis is most common. The muscles are toneless and the reflexes abolished. There is, however, no reaction of degeneration, and the outcome is usually complete recovery within a few weeks or months. Muscular atrophy sometimes is noted. It is not unlikely that some cases brought under this head really belong to the neuritides, or to myelitis, or are combinations of these with chorea.

**Diagnosis.**—The diagnosis of chorea is extremely simple when the motor symptoms are once developed. Difficulty arises usually through mistaking symptomatic choreoid movements in other diseases for true chorea. The escort of attending symptoms should differentiate these pseudochoreas. Here may be mentioned the *chytanic localizations* of metallic and toxic poisoning, of hysteria, and of multiple sclerosis. *Friedreich's ataxia*, *athetosis*, and *posthemiplegic chorea* have their cerebral signs. The two more sudden and more completely expressional or gesticulatory in character, showing their subconscious, purposive basis. *Huntington's chorea* almost invariably has a familial history, comes on after adult years, and is attended by progressive dementia. The *responderia* and the so-called *electrical choreas* present only a superficial resemblance to the chorea of Sydenham. The premonitory symptoms for a given case having been made out, their reappearance often enables the early diagnosis of recurrent attacks before motor symptoms attain prominence.

**Prognosis.**—In the great majority of cases chorea may be considered a self-limited disease of favorable prognosis. Complete recovery is the rule in children under ten years of age. In proportion as puberty is reached and adult years attained, the prognosis becomes more guarded both as to immediate results and the establishment of chronicity. Recurrent attacks, however, have a progressive tendency to earlier recovery. In a given case the intensity of the attack, the violence of the choreic motions, their generalization, the evidence of degeneracy in the individual, and a bad general physical state unfavorably modify the outlook for early recovery. The prognosis is also affected by the presence or development of cardiac involvement or the appearance of septic processes and high temperatures. The choreic status is of very grave significance, and usually terminates in death. Chorea in pregnancy also furnishes a high mortality for the mother and more often destroys the fetus.

**Treatment.**—In the treatment of chorea the constant evidence of muscular weakness and mental enfeeblement calls in decided tones for rest. Any emotional disturbance promptly aggravates the choreic movements. The child in ordinary cases must be taken from school and kept apart from the roughs and often from the gibes of its playmates, and for the most part in bed. A little effort to induce sleep, and frequent sponge-baths, gentle massage, and pleasant, quiet diversion will often, with rest, work great improvement in a few days. Toxics,



especially iron, are usually indicated, and attention to the constipation is always in order. The diet should be nutritious and easily assimilated, and contain plenty of fat in the form of cream and butter. It should be carefully controlled, as choreics are very prone to crave indigestible and objectionable articles. The use of drugs is of secondary importance. *Arsenic*, once in vogue, is valuable as a tonic in small doses, but usually harmful in large ones and only rarely efficacious against the chorea when pushed to the utmost limit of toleration. It is capable of producing all sorts of intestinal disturbance, and its protracted use may induce pigmentary changes in the skin or cause a multiple peripheral neuritis. It must, however, be admitted that in some cases a short, vigorous course of arsenic reaching ten to fifteen drops of Fowler's solution, three times daily, for a child six or seven years old, sometimes acts favorably. It may be commenced with a single drop and increased one drop a dose until the stomach rebels, which is usually at about twelve drops. It should then be stopped for a day and renewed at the final dose and slowly increased. It can rarely be used more than three weeks at a time with advantage. The use of quinine in large doses, as suggested by Wood, has in a series of cases in the writer's clinic failed to prove of any value.

In the severe cases absolute quiet and rest in bed or in a padded corner on the floor is required. Food must sometimes be given by the nasal or rectal tube and chloral must be used freely, associated with bromid if there is much delirium. Even morphia may be required, and strychnin to maintain the heart. Sulphonal, trional, exalgin, and nitipyrin have given help in some such cases. Small doses of apomorphin from the one two-hundredth to the one-twentieth by mouth or hypodermatic method, depending upon the severity of the case and the age of the patient, have been strongly recommended.<sup>1</sup> Hot baths and cold packs often are of distinct service. The conservation of strength and the support of the physical forces require most careful thought.

In the chorea of pregnancy it is seldom needful to terminate gestation, but if the motor storm is very violent and the mental features pronounced, or the physical state low, it may be indicated, especially as amelioration sometimes follows spontaneous abortion or delivery at term. Marinisco<sup>2</sup> recommends the intraspinal injection of from 3 to 5 c.c. of a 25 per cent. solution of magnesium sulphate in very severe and even in the pregnancy cases of chorea, to control the choreic motility. An antistreptococcus serum therapy has also found advocates.<sup>3</sup>

Complications, such as phlegmons, joint disease, and cardiac involvement, must be met on their indications. Recurrences should be anticipated and the parents should be taught the significance of sleeplessness, irritability, fidgetiness, capriciousness of conduct and appetite, and at once resume proper treatment. In girls this is particularly needed. In the pubescent period and later gestational experience, watchfulness should be exercised to maintain both the general health and the body-weight at a proper level.

<sup>1</sup> Tull, "N. Y. Med. Jour.," March 14, 1895.

<sup>2</sup> "Sem. med.," 1905.

<sup>3</sup> Mayr, "Wien. med. Woch.," 1908.

## CHAPTER V.

## \* MOTOR NEUROSES.

THE neuroses brought under the above heading have, in common, a preponderance of motor symptoms. Their grouping is entirely arbitrary and one of convenience.

## HUNTINGTON'S DISEASE.

In 1872 Huntington called marked attention to a number of families living in southeastern New York, who had for many years been under the continuous observation of his father and grandfather, both medical men. These people were afflicted with a family disease locally known as "megrins" or "megrums," and, owing to their peculiar motor difficulties, the patients were commonly called "shakers." The disease has been recognized in many parts of the world, and is variously designated *Huntington's chorea*, *chorea of the aged*, *family chorea*, *whisk hereditary chorea*, *chronic chorea*, and *chronic progressive chorea*. As early as 1850 See and Sanders described the condition under the name of chronic progressive chorea.<sup>1</sup> As it has no well-founded relation to Sydenham's chorea, the term *Huntington's disease* is here adopted as open to the least objection. The disorder presents an insidious onset in adult life, a marked hereditary character, and a well-defined tendency to mental deterioration and dementia.

**Etiology.**—The salient etiological feature of Huntington's disease is its *heredity*. It has been traced through five generations, and in a given family marks more victims than any other familial disorder, sometimes affecting as many as half of the entire number. It is transmitted about equally by males and females. In Huntington's series of cases it presented the peculiarity of never reappearing after once the hereditary chain was broken. Oppenheim, however, claims that intervening generations may escape the disease, but are prone to have epilepsy or other nervous disorders.<sup>2</sup> In contrast to true chorea it affects the male *sex* in greater proportion. Huot collected a series of cases embracing forty-four men and thirty-six women. It is also at variance with chorea in the factor of age. It usually appears from thirty to forty-five, but may develop at any later period. Exceptionally, it has been noted at puberty, never in childhood or infancy. Again, rheumatism in the personal and family antecedents is rare. In some instances fright or other mental trauma has appeared to induce it. Epilepsy is sometimes associated with it.

**Morbid Anatomy.**—It may be stated that no characteristic changes are recorded in the cases examined post mortem. They are those common to dementia in general. Thus Lannais and Puyet<sup>3</sup> in two cases found meningeal thickening, pachymeningitis, cerebral atrophy and compensatory hydrocephalus. The descending cord-tracts and the interlateral and cerebellar tracts were slightly sclerotic. The cortical layers presented a round-cell infiltration. C. A. Good<sup>4</sup> and Kattwinkel have

<sup>1</sup> "Archiv. f. Psychiatrie," Bd. 2, 8, 226.

<sup>2</sup> Lehrbuch, "Seltene Aufl.," Berlin, 1913, p. 1722.

<sup>3</sup> "Arch. de Neurol.," (ser. 1897).

<sup>4</sup> "Amer. Jour. of Insanity," July, 1900.



found in well-examined cases no evidence of inflammation, but widespread degenerative cellular changes throughout the cortex, most marked, however, in the frontal regions. Margulis<sup>3</sup> in 2 cases found great proliferation of neuroglia throughout the brain and cord, and feels justified in calling the disease a congenital chronic degenerative gliosis. He finds the cellular structures secondarily strangled by the proliferating glia.

**Symptoms.**—The motor symptoms commonly appear insidiously, but in some cases mental enfeeblement precedes. The face, the speech, or the gait may be first involved. Slow involuntary contractions modify the facial expression, cause a hand to start, a finger to move, or compel the feet to deviate from the intended direction. These motions at first are temporarily controllable, or cease on brief voluntary effort. Later, they are not under such control. The gait becomes progressively more erratic and uncertain, until, finally, it closely resembles that of drunkenness with the addition of gesticulatory movements of the arms and of facial contortions. The peculiar gestures, poses, and exaggerations of action in these cases are very prominent and often strangely at variance with the mental emotions actually in play. They are increased by embarrassment and emotion, but lessen in repose and subside during sleep. Though bearing a rough resemblance to the movements of chorea, they are more deliberate, gesticulatory, and of greater range. There is usually some muscular weakness, but no other modification of energy and none of sensation. Speech is thickened, dragging, and infiltrated with *h's* and *b's*, but not staccato or explosive, and may finally become impossible. In advanced cases the patient may become bedridden.

The mental state is one of progressive enfeeblement and depression. It is of slow onset and its natural goal is complete dementia. It may precede the motor symptoms, but usually follows them at a varying period. Thereafter the muscular and mental disabilities increase together to the end of life. A *dementia of ten to thirty years* is common and old age is often attained. Recoveries are unknown.

**Diagnosis.**—Huntington's disease encountered in a number of generations can offer no diagnostic difficulty. Originating *de novo*, it must be distinguished from chorea proper, from the *syndromes des lésés*, from double athetosis, and from the family ataxias. Chorea has its early mental symptoms, its cardiac lesions, its tendency to recover, and an absence of extreme mental degradation. Tics have a limited distribution, are much quicker in their rhythm or activity, and present no dementia unless occurring in idiosy. Athetosis is congenital, infantile, or postparalytic in development. The family ataxias have their eye symptoms, lack mental degeneracy, and commonly appear early in life.

Treatment has been futile.

#### MYOCLONIA.

The term *myoclonus* or *paramyoclonus* has been used to designate involuntary, unsystematized, arrhythmic, quick, muscular contractions, similar to those produced by an electric shock. They may be localized or disseminated and may embrace a muscle, a muscle group, or only a few muscular fibers. Under the general term myoclonia may be embraced

<sup>3</sup> *Deutsch. Zeit. f. Nervenk.,* Feb. 1913.

the *paroxysmus multiplex* of Friedreich, the *electric chorea* of Bergeron, Henoch, Paget, and the *fibrillary chorea* of Mervin. *Seizle chorea*, so called, is generally but the motor index of cortical degenerative changes. Myoclonia is sometimes combined with epilepsy, furnishing the "association disease" of myoclonus-epilepsy, to be described with epilepsy.

**Etiology.**—Practically nothing is known of the causation of myoclonia. Lundborg<sup>1</sup> has suggested that it bears some relation to the thyroid. Nervous heredity is commonly encountered, the male sex predominates, and adult age is the usual epoch, though Bergeron's form is most common in children. Overwork, fatigue, hunger, fear, transmutation, and cold have been considered excitants.

The nature of the disease is speculative, but the motor cells in cortex and cord are presumed to be at fault. A case in which muscular atrophy followed is thought to add force to this point of view so far as it relates to the cord. The morbid anatomy is practically unknown. Murri<sup>2</sup> found a chronic localized pachymeningitis with atrophy of the central cortex in one case.

**Symptoms.**—The onset may be sudden, following one of the inciting causes mentioned, or the motor spasms may insidiously develop. These are the essential features of the disease, which lacks sensory, trophic, dynamic, and electrical symptoms but usually presents increased tendon reflexes. The clonic contractions begin ordinarily in the lower extremities, and, as a rule, are bilateral, though not strictly symmetrical. They then involve the upper limbs, but commonly spare the face. The clonic contractions are instantaneous and involuntary, increased by emotion, but subject to some degree of voluntary control. They subside or remain in abeyance during voluntary use of the given muscles, at least for a few minutes. Depending upon their location, extent, and intensity, they may appear only as contracting muscular bundles, producing a linear elevation of the skin, or may cause a joint, a digit, or an entire extremity to start suddenly. Usually clonic, they may be repeated so rapidly as to produce a tonic, or even tetanic effect. One or all varieties may be observed in the same patient. The contractions are unequal, irregular, and arrhythmic. Sometimes they produce constant agitation; sometimes they come on in little attacks, with varying intervals of quiet. They may reach a rate of 60 to 100 a minute, and, as a rule, are more rapid the smaller the muscle affected. They cease during sleep, but in some instances may rouse the patient in the night. Permission, pinching, heat or cold, electric shocks, and the emotions tend to augment and recall them. The intellect is unaffected. The face, tongue, and trunk are exceptionally involved. The muscles of organic life and the sphincters escape.

When the disease begins insidiously, the contractions come on during repose and at long intervals, attracting little attention. They progressively increase in extent and vigor, and in a few weeks or months reach the period of complete development, which varies in duration in different cases, but is usually protracted many months. The tendency of the disease is then to subside slowly toward complete recovery, but often with remissions or later recurrences. Most cases finally recover.

**Diagnosis.** The diagnosis, owing to the rarity of the affection, is

<sup>1</sup> "Hygiea," 1900.

<sup>2</sup> "Arch. Ital. di Biol.," 1901.



seldom made. *Tics* are likely to be mistaken for myoclonia unless the case is carefully studied. These, however, are almost invariably first unilateral, and have a predilection for the face, whence they usually spread to the neck, arms, etc. They are distinctly purposive in character, and expressive or gesticulatory, demonstrating the subconscious basis on which they develop. Jacksonian fits are usually attended by sensory aura, mental disturbance, and major convulsions at some period of the disorder.

The prognosis is good.

Treatment has appeared to have little effect. The use of galvanism to spine and muscles is favored by some, and motor inhibitors, such as atropin, userin, valerian, hyoscin, and cocaine, have been employed with varying results. Commonly, the general physical condition requires appropriate attention.

#### DUPRE'S DISEASE.

In 1845 Dupré described a disease occurring in the malarial regions of Italy which he called *electrical chorea*. The name is unfortunate, as the condition is not allied to Sydenham's chorea, nor does it resemble it in any manner. This name has also been applied to several varieties of myoclonus, and to some hysterical manifestations, with equal disadvantage. The useful monograph is not yet determined, though many of these cases have been examined post mortem. In nearly all there is evidence of infection, such as pulmonary and splenic congestion, inflammation of the meninges, increase of cerebrospinal fluid, cerebral congestion, especially at the base, and softened foci in the cortex and great ganglia. In some earlier instances the cerebrospinal axis was reported of normal appearance. This statement may be received with some doubt. The disease has been attributed to everything, from malaria to typhus, and seems to be confined to Italy, and especially to Lombardy.

The onset is abrupt and marked by intense, continuous pains in the head, neck, and sometimes in the lumbar region. Shortly the extremities are seized with short, sharp spasms, recalling the electrical responses, and giving rise to the name "electrical chorea." They usually first appear in the upper extremities, especially in the hands and fingers, sometimes in the face, and are attended by painful sensations in the same locality. Gradually they spread into a hemiplegic or diplegic distribution, which is attained within a week or two. The twitchings occur in somewhat regular intervals, and frequently are accompanied by epileptoid convulsions, without loss of consciousness, which may take place several times in the twenty-four hours, and commonly leave partial traces behind them. Sensibility is not greatly affected, though sometimes hypersensitiveness may be easily provoked and excite the motor symptoms. Electrical reactions are said to be normal.

The disease usually grows progressively worse. The muscular spasms become almost continuous, the convulsions are rapidly repeated, and in from ten to one hundred and fifty days the disease ends in death in about ninety per cent. of all cases. The fatal termination is preceded by a condition of continuous epileptoid spasm, a sort of status, followed by coma, relaxation, and fatal exhaustion. Occasionally there are remissions in the progress of the disease. Treatment has been unavailing.

## PARKINSON'S DISEASE. PARALYSIS AGITANS.

In 1817 Parkinson gave a complete clinical description of a rather common disease, which he termed *shaking palsy*. It is generally known as *paralysis agitans*. As the usual weakness may be absent and the tremor may appear only late in the disease, the descriptive name is not always applicable. Generally, it is considered a neurosis, but accumulating material points to an organic basis for the malady, which will probably soon pass into its proper category. The disease is one of late-middle life, usually commencing locally, tending to hemiplegic and finally to diplegic distribution, and commonly marked by rigidity, tremor, and weakness of similar extent. Never fatal in itself, it lasts until death.

**Etiology.**—Parkinson's disease rarely commences before forty or after sixty-five years of age. Most commonly it appears at about fifty. That it is not essentially a senile disease is clearly shown by Willige,<sup>1</sup> who collated from the literature 47 cases under thirty years of age and a group of 12 under twenty. The male sex furnishes over two-thirds of all cases, and this proportion is uniform for all ages. A neuropathic heredity is commonly encountered. In exceptional instances the disease appears in several generations or in collateral family branches. Lundborg<sup>2</sup> has recorded 5 cases in one family, several other members being affected with myoclonia, both of which diseases he thinks related to disorders of thyroidal action. In another case he saw Parkinson's disease with myxoedema. Friedel<sup>3</sup> has also called attention to myxoedematous areas in paralysis agitans which are suggestive of a glandular factor. Among the alleged exciting causes, fear, anxiety, grief, and physical exhaustion have been named. In Paris and Strasburg during the sieges of the Franco-Prussian War and in the American Rebellion numerous cases developed under the combined influences of privation, prolonged anxiety, and sudden fears. Traumatism has appeared to incite it, and in such instances the tremor has sometimes appeared in the limb affected, sometimes even in the part of the limb directly injured. Krafft-Ebing<sup>4</sup> traced the disorder to trauma in 7 out of 110 cases. The possibility of hysteria in such cases is strong. Mental shock cannot be excluded in such accidents.

**Morbid Anatomy.**—Regarding the morbid anatomy of Parkinson's disease there is much diversity of opinion and observation. In typical cases Westphal, Berger, Charcot, and Gowers have found no abnormalities. Others have found changes in the encephalon, cord, and even in the muscles. Dulieu and Ketscher have noted retrograde changes in the motor cells of the anterior horns and sclerotic changes in the white tracts of the cord, recalling those due to senility. Rollich<sup>5</sup> in seven cases found the cord most affected. Small patches of sclerosis were found, mainly in the posterior columns, but some in the lateral tracts, and most frequently at the level of the cervical and lumbar enlargements. They originated from the vessels, and showed atrophy of nerve-fibers and increase in interstitial tissue. The process was an endo- or peri-arteritis, with extension to the surrounding parts. The cells of the anterior gray and Clarke's columns were almost always

<sup>1</sup> *Zutsche. f. d. ges. Neur. u. d. Psych.*, 1911.

<sup>2</sup> *Hygien.*, 1900.

<sup>3</sup> *Deut. Zst. f. Nervenh.*, April, 1896.

<sup>4</sup> *Wien. Klin. Woch.*, 1892.

<sup>5</sup> *Centralbl. f. allg. Path.*, Nov. 4, 1904.



pigmented. He recognized the changes as senile in part, but thinks they exceed it in degree. Dana has described a diffuse sclerous myelitis with vascular lesions and cellular atrophy in both cord and cortex. Mensel, Charcot, Bloch, and Marinesco in three cases have found tubercles in the peduncular region, and Brissaud, from a study of pseudobulbar palsies in connection with paralysis agitans, is inclined to locate the lesion in the peduncular territory. The monoplegic and hemiplegic cases indicate cerebral disorder and the involvement of the face and jaw-muscles is certainly due to supranuclear states. Gombier<sup>1</sup> has collated 24 cases examined by recent methods. In all there was decided uniformity of anatomical findings. These involved blood-vessels, neuroglia, and nerve-cells. There was proliferation of nuclei and thickening of vascular walls, increase of neuroglia about the blood-vessels, and patches of perivascular sclerosis, with pigmentation, degeneration, and atrophy of nerve-cells and nerve-fibers. The spinal cord was most affected, the changes being most marked in the gray matter and posterolateral portions of the lumbar and cervical enlargements, usually decreasing backward, and sometimes not appearing in the cerebration. Though resembling senile changes, the lesions were more intense and general arteriosclerosis was usually absent. Clinical resemblance to Wilson's disease (page 187) due to progressive degeneration of the lenticular nuclei and to other extrapyramidal cerebral lesions have drawn marked attention to the basal ganglia and particularly to the globus pallidus as is insisted upon by Ramsey Hunt. These ganglionic masses show in a marked degree the involutional changes. Fatty changes in the muscles are encountered in very old cases, and sometimes a peripheral nerve degeneration of a slight degree. Schief-fenlecker and Schultze<sup>2</sup> find the muscle-fibers, fibrille, and nerve quindles diseased, but no change in the nerves. Catola<sup>3</sup> has described changes in the muscles indicating a toxic chronic nodular myositis, and occasionally patients do complain of tender muscular thickenings. C. D. Camp<sup>4</sup> calls prominent attention to the parathyroids and suggests that their disease gives rise to the muscle changes through an auto-toxic effect. In 1897 the author treated many cases with desiccated parathyroids prepared in the Armour Laboratory, but without appreciable benefit.

**Symptoms.**—A typical case of Parkinson's disease presents a most striking picture. The patient trots into the room with short, reluctant steps, apparently following his center of gravity. The body is inclined forward, the neck extended and rigid, the elbows flexed and slightly abducted, bringing the hands, with their trembling fingers, to the level of the groins. The face is mask-like, the eyes bright and unblinking. The patient turns bodily, deliberately, and rigidly. He sits down



Fig. 101.—Parkinson's disease. (Illustration.)

<sup>1</sup> *Ann. Jour. Med. Sci.*, Dec., 1899.

<sup>2</sup> *Deut. Zei. f. Nervenk.*, Dec., 1903.

<sup>3</sup> *Riv. di patologia nerv. e ment.*, 1900. <sup>4</sup> *Jour. A. M. A.*, April 11, 1907.

slowly, with precaution, on the edge of the chair, always leaning forward, his shaking hands on his knees in constant motion. In the more advanced cases the patient drops rigidly or falls backward into the chair. Every change of position is studied and reluctant. We may take up the symptoms in detail.

The muscular rigidity is worthy of first attention, as it is the dominant motor phenomenon, causing the peculiar attitudes and postures, the immobile face, and the slowness of movement. It is the motor analogue of the mental inertia so common in this disease. It is nearly always present where the tremor exists, and may be highly developed without the tremor, or may first invade the parts that subsequently



Fig. 220.—Rigidity, attitude in parkinsonism.



Fig. 221.—Parkinsonism. Rigid facial expression.

tremble. Owing to the rigidity, the reaction-time is much increased. Dyle<sup>1</sup> calls specific attention to the fact that, while voluntary active movements are generally weak, the subjects of Parkinson's disease can oppose passive movements with practically normal strength, but passive motion is also sometimes impeded by the rigidity. There is some hypertonus, and the tendon reflexes are usually slightly increased. A foot-clonus is never encountered. There is a characteristic *facies*. The nasolabial folds and lines of expression tend to disappear.

The face becomes smooth. The frown may retain its cross-wrinkles through enforced elevation requisite for forward vision, if the body and head be bowed. The eyes are widely opened and rarely wink. The ocular globes tend to remain fixed so that the patient, in order to change the direction of vision, ordinarily turns bodily with the neck held rigid. Movements of ocular association and accommodation are impeded by this rigidity, and the patient shows little or no facial variation for any of the emotions that may play behind this mask. Speech is modified. It becomes monotonous and deliberate. The patient



Fig. 222.—Rigidity in parkinsonism, showing propulsion.

<sup>1</sup> "L'Encéphale," 1909.



hesitates, but, once started, hurries his sentences and stops abruptly as if relieved to be through. He likes monosyllables and may become extremely taciturn. In some cases tremor and rigidity of the vocal cords have been observed by means of the laryngoscope. In some instances there is an excessive secretion of saliva. In others true bulbar symptoms are added with indications of bulbar-glossopharyngeal palsy.<sup>1</sup>

In the neck all the muscles are involved, giving an appearance of stiff-neck or rheumatic torticollis, but the face is always held to the middle line, the chin commonly somewhat advanced. The body is bent forward throughout its length and also at the hips. There is everywhere a preponderance of flexor positions, as a rule, but in very exceptional cases the neck and body may be bent backward. The arms, naturally drooping forward, are flexed at the elbows, which are slightly separated from the sides. In making passive extension of the elbow the muscles will often be felt to yield in an interrupted manner, communicating several jerky interruptions to the smooth excursion of the member. This *rag-wheel-synopsis* is quite significant and may be sometimes discovered very early in the course of the disease. The hands may be partly flexed or extended at the wrist, but the fingers are always held more or less in flexion. A position similar to that of holding a pen is common, or the hand may be partially closed. The digits frequently deviate to the ulnar side of the hand, as in rheumatoid conditions, and these may also be present. In the lower extremity the stiffness is less marked, but in advanced cases causes a knee-spring attitude and gait.

The gait of Parkinsonians is strikingly peculiar. When the patient rises from the chair, he hesitates a moment as if to take aim, and starts ahead in a direct line, his laggard legs trotting to keep up with the forward-leaning body. In some instances the patient is strongly impelled forward, and can only arrest himself by running into objects or passers-by. There may or may not be an actual tendency to fall forward, or propension, but in some cases, if the body be started backward, sideways, or forward by a push, the direction is unmaintained for a few or many steps,

*It is pleasant.*

*Rapids Iowa*

Fig. 205.—Handwriting in Parkinson's Disease, with normal style below.

giving rise to the terms *intersuspension* and *retrogression*, and these may occur spontaneously on getting up or in attempting to stop while advancing. As described by Stewart,<sup>2</sup> in advanced cases the method of getting into bed is characteristic. The patient climbs on to the bed,

<sup>1</sup> Brauer, "Neurolog. Centralbl.," Nov. 1, 1901.

<sup>2</sup> "Lancet," Nov. 11, 1898.

stands up, and, bending down very slowly, grasps the rail at the foot-board. Holding firmly to the headstead he slowly sits down and then falls or rolls backward into the recumbent posture.

The trembling in shaking palsy may appear after rigidity has developed or at the same time. It usually commences in one hand and arm and then invades the lower extremity, subsequently appearing in the opposite arm and finally in the opposite leg. In some cases it is bilateral from the start, but commonly it is more marked on one side than on the other, and may be monoplegic or more often hemiplegic for several years, eventually showing a tendency to diplegic distribution. Often it is steadily preceded in its advance by the rigidity, or the rigidity may be generalized and of long standing before tremor appears in the hands. It affects the distal portion of the extremities most. In the hands, where it customarily first appears, and is most developed, it causes a rhythmical, alternating flexion and extension of the fingers, mainly at the metacarpal joints. The tremor may be limited to the index and thumb, or affect the interossei most, causing rolling of the fingers upon their long axes. The patient appears to be constantly rolling some small object, or a pill or a pencil, between his fingers and the opposed thumb. Sometimes flexion and extension of the wrist are added, and very rarely we encounter movements of pronation and supination. As a rule, the arm and shoulders are unaffected. In the lower extremity the tremor predominates at the ankle, causing the foot to drum on the floor as if with clonus. The toes are less evidently involved by tremor, but many patients complain of flexor cramping of the toes as a very early condition. This usually comes on while walking, and may bring the patient to a momentary standstill. The muscles of the thigh often participate in the tremor. The muscles on the back of the neck, shoulders, and dorsum of the body are least affected. The abdominal muscles apparently escape. In the great majority of cases the head does not tremble, or only does so by movements communicated to it from a distance. In very rare cases, however, there is a rhythmical nodding, shaking, or rotation of the head that may persist even when the patient is recumbent. The eyelids exceptionally are affected, while the lips and lower jaw not rarely show a tremor synchronous with that in the hands.

The great peculiarity of this tremor is its usual occurrence while the patient is at rest, during repose, and while the parts are supported. In a minority of cases, however, and in other cases at an early period, the tremor is *intentional*—that is, analogous to the tremor of multiple sclerosis. The tremor ceases during sleep, and usually it subsides immediately on voluntary motion. In early cases, and particularly in cases presenting marked antecedent rigidity, the tremor may only appear on voluntary and sustained motion, as in reaching to the back of the neck or into a distant pocket, and must be carefully sought. The tremor is a slow one, of from four to eight oscillations in a second. Usually the movements are more rapid if of limited extent, as when confined to the fingers, and grow slower as they involve larger muscles. When both upper or all four extremities are involved by the tremor, there is a practical synchronous uniformity of rhythm at all points. The character of the tremor shows distinctly in the *handwriting*. The letters are formed slowly and are of fair proportions, but all the lines are tremulous, both



apostrophes and stems. The writing tends to become cramped and small. Sometimes a lens is required to detect the tremor thus graphically demonstrated.

The palsy never reaches a complete degree, and the paresis may be extremely slight. While patients may bitterly complain of a feeling of weakness and stiffness, they often show a normal amount of strength, even when the rigidity and tremor are very well developed. In advanced cases, however, there is customarily some weakness, and this may even be extreme. These patients, as a rule, are loath to make exertion of any sort.

**Sensory Disturbances.**—The general sensibility is practically objectively intact, but Palmieri and Armand<sup>1</sup> insist that the parts affected by tremor show decided hypalgæsia, most intense in the distal portions of the limbs and gradually fading off toward the trunk. Karpis,<sup>2</sup> in a study of 103 cases, never found objectively disturbed sensation without tremor, and subjective sensory complaints were noted in but 35 per cent. Parkinsonians frequently complain of subjective feelings of heat, more rarely of cold, and often of dull aches and indescribable discomfort in the affected limbs. It is not rare for Parkinsonians to complain of considerable pain in the early stages of the disease similar to rheumatic pains in joints and muscles. The heat sensations may be accompanied by *vasomotor disturbances*, showing itself in elevated local temperature, in profuse sweats, and in flushing. Sometimes patients seek cool rooms and throw off heavy clothing and bed-covering, even in winter. In the majority of cases complaining of heat the surface temperature is actually increased. In some cases areas of *honey skin* are encountered on the face or body, a condition suggestive of myxodema and of scleroderma. Often there is great restlessness, apparently due to the discomfort arising from muscular rigidity. The limbs may be frequently moved or the patient insists upon the limbs being rubbed and moved about every few minutes. Muscular atrophy only appears in advanced cases, but even then is not extreme. There are no electrical changes or sphincter weakness. As this is a disease of the involutional period of life, we may find all the disturbances of senility as coincidental accessories.

The mental state is likely to be mistaken for one of dementia, but, as a rule, these patients enjoy all their mental powers. There is, however, the same inertia in the mental processes that marks the muscular state. They shun exertion, are chary of their thoughts, talk little, appear indifferent, and often require the incentive of strange faces or extraordinary circumstances to arouse them to a show of mental activity. This, taken with their inexpressive faces, is easily misleading.

**Course.**—The disease is essentially chronic and progressive. The onset, insidious, as a rule, may be abrupt, following some mental or physical storm. The duration is from ten to forty years. In the extremely protracted cases the patients become more and more helpless, fall into a senile dementia, and die from intercurrent disease, usually pneumonia. Faciès have been described, but they are usually limited and undeveloped cases. Thus, the *metoplegic* and *hemiplegic* forms,

<sup>1</sup> "Clinica Med. Ital.," No. 6.

<sup>2</sup> "Laksh. & Psych. u. Neurol.," 1900.

the form without tremor, the form without rigidity, and the form showing extension are aimed. The *prognosis* is bad, but there may be remissions.

**Diagnosis.**—In typical cases the diagnosis is made at a glance. In early and undeveloped cases Parkinson's disease may be mistaken for *posthemiplegic trembling*, but lacks the history of a stroke. *Senile and family trembling* usually first affect the head and do not prevent the faces and rigidities, but intermediate cases may be found and both may coexist. *Multiple sclerosis* has its increased reflexes and intention tremor; *hysteria*, its stigmata. When beginning in the right hand and not marked by continuous tremor it has been mistaken for *writer's cramp*, but the disability is equal for all voluntary use and not limited to occupational activity.

**Treatment.**—If seen early, the case should be treated as one of cerebral arteriosclerosis (see p. 235). In several instances this plan of treatment has seemed to retard the development of the disease. Symptomatic medication is practically useless. Opium, hyoscin, and cannabis indica, given freely, temporarily control the tremor, but at the expense of the general health and welfare of continuously employed. Many patients find hyoscin useful as an occasional help to meet some social or business requirement. Massage, electricity, and strychnin give a little help for the time being in some cases. Persistent exercises,—both passive and active,—Swedish joint-movements, and gentle muscle kneading, if intelligently carried out for a long period of time, certainly benefit these patients in the earlier stages. Especial attention must be given to build up the extensor muscle groups of the limbs and trunk in order to overcome the natural tendency to flexed positions, and full passive movements to the same end should be perseveringly employed. For the same reason exercises or electrical treatments tending to strengthen or irritate the flexor muscles are to be avoided. Mental and physical fatigue must be shunned. Charcot noticed that the vibrations experienced in carriage and car-riding mitigated the tremor, and treated many cases by means of a jolting or vibrating chair with temporary benefit.

#### THOMSEN'S DISEASE (MYOTONIA).

In 1876 Thomsen, himself subject to the disease, fully described a muscular condition later called *myotonic congenita*, *family myotonia*, etc. It is a disorder manifest in the voluntary muscles, which show a stiffness and rigidity upon attempted use after a period of repose, and certain peculiarities of mechanical and electrical irritability. It is a rare disease, numbering not more than a hundred recorded cases.

**Etiology.**—The salient etiological feature of the infirmity is heredity. Most of the known cases have been in family groups, sometimes extending over several generations and through several collateral branches. Occasionally the disorder has passed over a generation and again appeared. With and without direct heredity the family history is commonly surcharged with neuroses and psychoses. Males are apparently the more commonly affected. Von Bechterew<sup>2</sup> suggests that a self-poisoning or auto-intoxication is active in this condition, and Jarely<sup>3</sup>

<sup>1</sup> "Neural CentralB," Vol., 1900.

<sup>2</sup> "Zentr. Nerv. und Ment. Dis.," July, 1905.



looks upon the disease "as due to an embryonal developmental disorder of the nerve-cells, consisting in the more or less diminished resistance of the cells to the influence of toxic processes." Johnson and Marshall<sup>1</sup> offer the suggestion of an increased resistance to the passage of impulses in the synapses and found that small doses of strychnin which is known to act on the synapses of the central nervous system relieved the myotonic features. The disease is also related to the myopathies, as shown by the muscular contours and the occasional appearance of atrophy.<sup>2</sup> Paessler, Pels, Rossolino, Batten and Gibb,<sup>3</sup> and others have recorded upward of thirty cases in which the atrophy was the predominant condition. These are commonly designated as cases of *myotonic atrophie*, and constitute a marked link between the myopathies and the myotonias.

**Morbid Anatomy.**—Various observers, upon examination of excised fragments of the affected muscles, have recognized a hypertrophy of the protoplasm and nuclei and a deficiency of striation in the enlarged muscle-fibers, usually with slight but insignificant increase of interstitial tissue. This constitutes, according to Delage, a persistence of embryonal conditions. Jacoby insists that this appearance is an artifact that is not found if the tissue removed during life is not allowed to contract. Bates and Marinaccio have noted deformity or maldevelopment of the terminal motor-nerve plaques. Schiefferdecker asserts that there is a distinct disorder of the sarcoplasm and disease of the muscle fibrils. The hypertrophy appears to be secondary and the increase of nuclei is proportional to it. In the first recorded autopsy Dujérine and Sottas<sup>4</sup> found no changes in medulla, cord, or peripheral nerves. It is as yet impossible to say whether we have to deal with a pure myopathy, a trophoneurosis, or a congenital defect in the trophic and motor apparatus of the cord.

**Symptoms.**—The awkwardness caused by the fixity of the muscles upon attempted use is noticed in infancy, or may appear at any time up to the twentieth year, or perhaps even later. In typical cases, when the subject desires to execute some movement, a more or less marked and prolonged contraction fixes the muscles in question. This gradually subsides and the movement is accomplished. Repetitions of the spasm occur progressively with less force and duration for the particular action, and finally cease to appear, but any change in the character or even in the rhythm of the movements may reinstate the muscular fixation. Thus, in rising from a chair the legs and thighs are held rigidly. Once erect, the first step is impeded, the second less so, and finally steps are taken with natural ease, but a halt, a sharp turn, or even a change of speed may again set up the spasm. All the voluntary muscles may be affected, even to those of the thorax, eye, and tongue, but usually the myotonia is most marked in the lower extremities, and in some cases the face and upper extremities escape. In marked contrast to the retardation of voluntary muscular action is the prompt response of the same muscles to reflex excitation which would indicate a block somewhere

<sup>1</sup>"Quart. Jour. Med.," 1915, vol. p. 111.

<sup>2</sup>Bersheim, "Allg. med. Centralbl.," No. 14, 1890. <sup>3</sup>"Brain," 1909, p. 187.

<sup>4</sup>"Rev. de Méd.," Mar., 1893.

in the upper neuronic field. The *congenital perimyositis* of Eulenberg, in which symmetrical groups of muscles are affected mainly on direct exposure to cold, appears to be a limited form of Thomsen's disease. Both varieties have been observed in the same patient by Bernhardt.<sup>1</sup> The sphincters and unstriated muscles escape, and in the case mentioned by Eulenberg the heart-muscle was normal. Gaping, sneezing, cold, wet, fatigue, and emotional excitement provoke the spasms, while warmth, moderate exercise, repose, and quietude diminish their intensity. They are in some measure relative in intensity to the vigor of the attempted movement.

Commonly, the affected muscles are of unusual firmness and increased bulk, but of lessened power, giving an appearance of athletic development at variance with the actual weakness, in many cases suggesting a pseudohypertrophy. They are perfectly supple to passive movements. The reflexes are normal, but a tap on the tendon is likely to produce a spasm in the anterior femoral group, modifying the usual response. Electrical and mechanical excitations of the motor nerve-trunks produce perfectly normal responses, or they are, if anything, somewhat diminished. In the *muscles* it is very different. A slight blow, as with a percussion hammer, produces a persisting rest from localized muscular swelling or myoklema. The galvanic current produces sluggish, prolonged contractions upon closure almost equally with either pole. Anodal or cathodal closing tetanus may often be secured with the continuous passage of five to ten milliamperes of current, and anodal opening tetanus is not infrequently observed. The strong faradic current produces undulatory contractions in many muscles, and these sometimes attend the passage of the continuous current. Repeated electrical or mechanical stimulation of the muscles, like voluntary use, gradually exhausts the myotonic responses.

As varieties, Jacoby would limit the term *congenital myotonia* to cases (1) presenting a hereditary etiology either as a direct transfer from the ascendant, or by inherited disposition; (2) manifesting the myotonic disorder of movement—namely, intention spasm; (3) showing the myotonic reaction, which he describes as made up of normal mechanical and faradic excitability of the nerves and increased mechanical and faradic excitability of the muscles, anodal and cathodal contractions being equal and the response always being tonic and prolonged; (4) persisting hypertrophy of the enlarged muscles; and (5) absence of symptoms pointing to gross involvement of the nervous system. Other varieties of myotonia he would denominate *myotonia acquisita*, or describing those acquired subsequent to birth, and *myotonia transitoria* for the cases due to exposure to cold, etc. Lamouroux<sup>2</sup> has noted an association of progressive muscular atrophy and myotonia in an individual of apparently excessive muscular endowment and finds that about a dozen such cases have been reported.

*Paralytic disorders* are frequently associated, but not necessarily present. The malady, once developed, tends to persist for life, which it does not

<sup>1</sup> *Lancet*.

<sup>2</sup> "Nour. Journ. de la Salpêtr.," Nov., 1900. Also W. Fumagalli, "Deutsche Zeitsch. für Nervenheilkd.," 1907.



abridge. It is an incurable infirmity, but sometimes shows arrest or amelioration.

**Diagnosis.**—The myoclonus and myotonic electrical reactions, taken with the intention spasm, if the term may be used, make the diagnosis easy. *Tetany* has its distinctive signs in the phenomena of Trousseau and Chvostek. *Pseudohypertrophic paralysis* has peculiar deformities, contractures, and weakness without intention cramps. It subsequently shows atrophies and has no myotonic reactions.

**Treatment.**—The causes which provoke the myotonic cramps, such as overexertion, fatigue, exposure to cold, and excitement of all sorts, must be avoided. Massage, reasonable exercise, electric baths, and cerebral galvanization have been recommended.<sup>1</sup> A careful search for toxic factors should be made, and conditions known to be attended by them should be corrected. The continuous or interrupted use of strychnin may be of some benefit.

#### FAMILY PERIODIC PARALYSIS.

There is a form of periodic limp paraplegia that may be designated *familial periodic paralysis*. It is characterized typically by recurring attacks of pure flaccid motor palsy, most pronounced in the lower extremities, marked by diminished reflexes and lessened electrical and mechanical muscular excitability. The intervals are those of ordinary health, and there is a decided family and hereditary tendency.

The first definite outline of this condition was given by Westphal, in 1885. Additional material has been contributed by Göbbel, Oppenheim, Bernhardt, Hirsch, and other German observers, and by Barr, Taylor, Mitchell, Putnam, and Crafts in this country. Oddi and Aubert<sup>2</sup> were able to collect 64 published cases.

**Etiology.**—*Hereditary* is most apparent. Taylor noted that thirty-five cases had been reported in three families, nineteen in one of them, and in two instances it had appeared in five consecutive generations, descending through both sexes, which are about equally represented. Nearly all known cases presented attacks before the age of twenty-five, the great majority in youth, rarely, however, before the tenth year of life. The leading causes of the attacks have been exertion, fatigue, and mental strains. Göbbel, in 1890, suggested that there was an *arthritic* causation acting upon an inherited vulnerability of motor nerve-cells, and Crafts<sup>3</sup> and Lewin have been able to isolate an extractive from the freys passed immediately after an attack which produced temporary paralysis when injected into rabbits and guinea-pigs. The amount of urea excreted during the attack also appears to be diminished. These results have not been confirmed.<sup>4</sup> Somewhat analogous attacks due to malarial infection have been cured by quinin. The muscular contours, and in Bernhardt's case atrophy of the thenar eminences and continued muscular weakness, imply a relationship to the progressive muscular atrophies and to the myotonias. Examination of muscular fibers taken from the living subject also indicates a similar kindred. Bernstein suggests<sup>5</sup> that this disorder may be related to, if not identical with,

<sup>1</sup> W. von Nothmann, "Neurolog. Centralbl.," Nov., 1897.

<sup>2</sup> Arch. gén. de Méd., 1902.

<sup>3</sup> "Am. Ass. Med. Sciences," Jan., 1900.

<sup>4</sup> Mitchell, Fleisser, Fittell, "Brain," 1902. Singer, "Brain," 1901.

<sup>5</sup> Deutsch. Zeitschr. f. Nervenk., Nov., 1908.

epilepsy. His reported case was epileptic before the paralytic attacks appeared, and apparently substituted them, and there also was an epileptic sister. Schachnowicz<sup>1</sup> has reported an instance in which the patient's father was similarly affected, and his brother was an epileptic. After many years of periodical paralysis these attacks subsided and epileptic attacks occurred.

**Symptoms.**—The attacks come on rather slowly within a few hours, usually at night, or during sleep. They are sometimes preceded by a feeling of weariness, numbness, formication, sweating, heat, desire to urinate, headache, backache, rapid pulse, coldness of the legs, etc., in various cases, but all prodromata may be lacking. In some instances the attacks begin as a migraine or alternate with migraine.<sup>2</sup> The lower extremities are uniformly most affected, and the paralysis may be limited to them, but in other instances involves every skeletal muscle except those controlled by the cranial nerves, so that the patient is inert from the chin downward. A facial weakness has been observed in one case. Even the voice and respiratory efforts may be weak, sneezing and coughing impossible, and the heart has in certain instances been found dilated and with evidence of mitral insufficiency, both disappearing with the attack. Such attacks last from an hour to a week and tend to uniformity in a given case. The paralysis recedes in the reverse order of invasion. Attacks may come daily, weekly, or once in several years.

During the attack electrical responses in nerve and muscle and the tendon reflexes are lessened or completely abolished, but there is neither reaction of degeneration nor splintastic incompetence. Mechanical excitability of the muscles and nerves also usually disappears. Bornstein<sup>3</sup> reports a case in which the tendon reflexes persisted and at times were increased during the attacks, though the electrical excitability was diminished or even abolished. The mind and general sensation and the special senses are unimpaired. The attack subsides about as rapidly as it develops, and in the intervals the health is usually perfect. Indeed, many of these patients are described as unusually robust and commonly very muscular. The muscular contours at times have even suggested the myopathic family disorders.

**Diagnosis.**—The diagnosis in familial cases should be easily made. In sporadic instances the behavior of the reflexes, electrical and mechanical responses, periodic recurrence and absence of mental and sensory disturbances, should be sufficiently definite to distinguish the condition from *hysteria*, with which it has been confounded. A first attack may suggest *Lambert's paralysis*, but again reliance may be placed on the quantitative electrical changes.

**Prognosis and Treatment.**—Thus far no treatment has been of much service, but these conditions, such as fatigue, which appear capable of inducing attacks in given instances must be avoided. In the trigeminal cases bromid and caffeine have proven useful. Holtzappel<sup>4</sup> asserts that he has derived great benefit by the administration of bromid of potassium with caffeine citrate both in aborting the attacks and

<sup>1</sup> "Wien. Med. Wochenschr.," 1882.

<sup>2</sup> "Holtzappel," "Ann. Med.," April 30, 1904.

<sup>3</sup> "Deutsche Zeitschr. f. Nervenk., Band 22, S. 407.

<sup>4</sup> "Jour. Amer. Med. Assoc.," Oct. 21, 1905.



entailing those already established. The tendency is for the disease to endure for life without compromising it. A further knowledge of the toxic states may furnish the key both to the pathology and treatment of this rare disease.

#### FAMILY TREMOR.

A tremulous condition, particularly of the hands, but also in some instances involving the head, face, and tongue, closely resembling the tremors of multiple sclerosis, scurvy, alcohol, and mercury, is encountered as a family trait. It may be traced through several or many generations, and affects a large proportion of the family members, appearing before forty and usually before twenty years of age.

The majority of such families are markedly asthetic, and, according to Raymond, the tremor may be considered as a stigma of degeneracy. In character the tremor is usually fine and rapid. It is accentuated by effort, fatigue, and emotion, seldomly does not occur during rest, but generally closely resembles the intention tremor of multiple sclerosis.

### CHAPTER VI.

#### FATIGUE NEUROSES.

MANY occupations requiring the constant repetition of certain precise muscular movements may, eventually, through overuse and fatigue, give rise to disturbances of muscular control for the maneuver in question. The condition may be manifest as pain, tremor, weakness, or cramp, but usually these are variously combined. This group of motor disturbances is also called *occupation spasms* or *occupative neuroses*. Many of them are described under terms indicating their particular avocational association, as *scrivers' palsy*, *piano-players' cramp*, *seamstresses' spasm*, etc. We may take *writers' cramp*, the most commonly encountered one, as a type, and then only an enumeration of the other forms will be needed. The more delicate and highly differentiated the functional movements, the more readily does their repeated overuse set up this inhibitory condition. No definite anatomical changes have been found, but it is probable that improved methods will demonstrate morphological alteration of the nuclear gray. Vigouroux<sup>1</sup> claims to have found changes in nerves and muscles in many cases, and supposes them to be present in all.

#### WRITERS' CRAMP.

Writers' cramp is variously known as *scrivers' palsy*, *grapho-spasms*, *megagraphia*, *chirospasm*, etc.

**Etiology.**—A neuropathic heredity and a neurotic make-up are very common among sufferers from writers' cramp. The scuriosis has exceptionally been noted in brothers and in parents and children, but, ordinarily, there is merely a transmitted nervous taint constituting a

<sup>1</sup> "Progrès Médical" quoted in "Amer. Medical-Surg. Bulletin," Jan. 15, 1897.

tendency to the development of the disorder under the provocation of overuse of a certain functional group of muscular movements. It is much more common in the male than in the female sex, perhaps owing to the greater proportion of men engaged with the pen. It most commonly develops between twenty and fifty years of age, being very rare before and after these extremes. Its greatest incidence is between twenty-five and thirty-five. It is very likely to appear during periods of physical or mental strain, especially after protracted anxiety arising from any cause. Occasionally, some local injury to the hand or arm which entails additional difficulty in the mechanical process of writing



FIG. 203.—Transverse cell from posterior spinal ganglion of cat (from Boddy, after Take).



FIG. 204.—Cell from posterior spinal ganglion of cat. Effect of stimulation (from Boddy, after Take).

may serve to provoke it. Neuritis, neuralgia, and hemiplegia affecting the writing arm have been followed by the neurosis. The principal inciting cause of the trouble is *excessive writing in a bad manner*—that is, in any style that depends upon the employment of the small muscles of the hand, wrist, or forearm in chief, and in which the writing movements are not made entirely from the shoulder. Thus, writers' cramp is practically unknown among stenographers, in spite of the rapidity and tediousness of their work, owing to the fact that the characters employed are best made by the freeland method customarily employed. Gowers, indeed, encountered a stenographer who could write shorthand readily, while ordinary script caused a spasm.

**Pathology.**—Numerous theories have been advanced regarding the pathology, and the seat of the disease has been variously placed in the muscles, in the nerves, in the spinal centers, in the cerebellum, and in the cortex. We can, at once, rule out the muscle and the nerve as initial loci, if the muscular control for movements not of the particular occupational variety are fully, promptly, and normally executed. It is impossible to conceive of a peripheral lesion which would disturb only a certain purposive function, leaving others intact. On this point W. E. Paul<sup>1</sup> insists that a careful scrutiny will discover a certain amount of defect for all activities of the involved muscle groups. He attributes etiological importance to the traumatic effect of repeated muscular contractions upon nerves, nerve-endings, and muscle tissues. A her-

<sup>1</sup> "Jour. Amer. Med. Assoc.," 1911.



grade neuritis is undoubtedly present in some cases, and Vigoureaux, already quoted, thinks it invariably present. In most all cases the motor function at fault is one to which the motor apparatus has become trained and habituated. In some degree it is automatic and subconsciously. Such acts are generally supposed to be largely subcortical. Another factor is interposed by the fatigue element. Hodge has clearly demonstrated the changes in motor cells resulting from physiological fatigue. In the occupation neuroses it is at least supposable that the fatigue may overpass the limit of recuperation. Well-authenticated cases in which muscular atrophy without sensory defects has succeeded the spasm would indicate rather conclusively that such was the case, that the motor cells of the cord were at fault, and that their trophic powers were finally involved. Sensory and motor symptoms, functionally grouped, would also point to the cord, but it is impossible as yet to exclude the superior cortical centers. In the neuritic cases tenderness and sensory faults are of characteristic anatomical distribution.

**Symptoms.**—The onset of writers' cramp is commonly insidious. It is noticed after writing a considerable time that there is sensory discomfort or motor difficulty. The hand aches, feels numb or weak, or may become cramped or tremulous. After a moment's rest and a little rubbing writing may be resumed for a time, when the trouble reappears and gradually less and less work is required to induce the disabling condition, until in extreme cases merely putting the hand in the writing position may cause it. Several forms have been described depending upon the major symptom. Thus we have the *spastic, tremulous, neuralgic, and paralytic varieties*; but they are very rarely pure, most cases showing a combination of sensory and motor features. Ordinarily, the cramp is painful not from the muscular contraction, but because the parts are affected with neuralgic pains that are occasioned only by the act of writing. In some instances they are so severe as to alone attract the patient's attention and inhibit the act of writing. In other and much rarer cases a feeling of weakness appears to prevent the use of the pen, but it is usually associated with pain. Tremor may be the principal feature, and is practically a manifestation of weakness.

**Motor Disorders.**—In the case, Fig. 243, illustrated from photographs, the plan of holding the pen between the index and middle fingers was adopted when spasm first developed, and for a time permitted a continuation of clerical work. When seen later, the hand would begin to cramp after half a dozen words were written, as shown in the second cut, and in a moment the spasm would become violent, the pen would fall, and the fingers and thumb would be extended as shown in the third cut. This was an aggravated case. Commonly, as soon as the spasm causes uncertainty in writing the act is stopped and the spasm subsides. It may cause a great change in the writing, which tends to become cramped and full of uncertainties and zigzags. The pen is forcibly jerked and thrust in various directions. At first, by steadying the writing hand with the other, and adopting a thick penholder or one fitted with a ball held in the palm of the hand or with rings against which the fingers are pressed in extension, writing may be continued for





a time, but usually the spasm reappears. If the other hand be then used, it is not uncommon for the spasm to appear on that side after a few months, though this is not invariably the case. When the spasm affects the second hand, it may also cause the muscles of the first hand to



Fig. 242.—Writers' cramp (from photographs): 1, Pen held between fingers at first spasm; 2, spasm beginning to develop and subside; 3, fully developed spasm, throwing of the finger except small and ring into extension and causing the pen to fall.

contract. The spasm may also spread to the muscles of the forearm and even to the arm and shoulder.

In cases of moderate severity the spasm may be occasioned only by the act of writing, but sometimes, and in severe cases usually, other acts requiring precision in finger movements also call it forth. In telegraphers' cramp, writing with the Morse key or the pen often alike causes the spasm; musicians may experience the spasm both in instrumental manipulations and in writing; seamstresses by using the needle or the scissors, etc.

The power of the hand for coarse muscular efforts may be unimpaired, but in cases of long standing some weakness in the muscles most concerned is commonly found, and in such cases slight wasting may occur. In rare instances, when the use of the affected member is pushed in spite of the disability, permanent atrophy may result. In figure 244 is shown the hand of a seamstress in which local pain and spasm were finally followed by atrophy, the reaction of degeneration, and fibrillary twitching limited mainly to the radial half of the hand and ulnar border of the forearm, namely, to the muscles chiefly concerned in the use of needles and scissors. The electrical responses in muscles and nerves may be normal, but in protracted cases there is usually an increase and finally a decrease of excitability, and, as in the rare case illustrated below, the reaction of degeneration may be present.

**Sensory Disorders.**—The sensory disturbance is sometimes described as one of great fatigue, or there may be pain and tenderness. Tenderness along the course of the nerves is not uncommon. In some cases pain is located in the small bones and joints of the wrist or fingers. It may only occur on attempts to write, and it may or may not be preceded by spasm. In certain cases it extends to the arm, axilla, and shoulder. Such paresthesias as "pins and needles," tingling, ache, "tired feeling," etc., are often complained of, and may similarly extend up the arm. Actual hyperesthesia or anesthesia are encountered only when neuritis is associated.

**Course.**—Commonly insidious in onset, writers' cramp may develop with some suddenness, after an emotional, moral, or physical storm, and

tends to progress so long as writing is persistently attempted. If the opposite hand be trained, it usually also becomes the seat of cramp, which is likely to increase more rapidly than on the side first affected. If writing be completely abjured, the cramp gradually becomes less and



FIG. 244.—Flexing of muscles in a severe case of nonstrict cramp.

less, and may entirely cease after a few months or a year, but is very likely to return if continuous writing is again taken up. The tendency of the cramp, pains, and parasthetic sensations to persist and increase if the writing occupation is continued is pronounced, and there is danger of passing the condition into one of organic changes, with atrophy and permanent palsy.

**Diagnosis.**—The diagnosis is usually made by the patient, and nearly every large clerical force or telegraph office furnishes cases. *Myoclonia* and *hemioplegia* from organic brain disease are sometimes mistaken, as the patient's attention is first attracted by his clumsiness in writing. *Myoclonic* *poly*, *neuralgia*, and *rheumatic* pains are occasionally mistaken for writers' cramp, but in all these conditions the symptoms are persistent and not alone provoked by writing. Spasm, usually present in writers' cramp from the first, is also absent. *Hysteria* may present limited tremor and motor difficulty, but the disability is not confined to writing, and sensory stigmata are added. *Paralysis agitans* beginning in the hand may be confounded with writers' cramp, but the tremor, rigidity, and difficulty of using the hand for all other complicated or difficult purposes are not marked in occupational neuroses.

**Prognosis.**—The prognosis is very unfavorable if writing be persisted in. If the patient can completely abstain from the use of the pen or similar writing implements, the spasm tends to subside and disappear unless the case is of long standing and so aggravated that all sorts of finger use induce the cramp. The neuralgic cases are somewhat more easily managed than the cramp variety if prolonged rest can be secured. The presence of any removable diathetic disability, as *lithemia*, favorably modifies the outlook, and the termination of general depression, physical or mental, may lead to marked improvement in the spasmodic affection. The tendency to recurrence and to extension has already been noted. It is rare for a patient, even after years of rest, to be able to return to writing occupations without a reappearance of the neurosis.

**Treatment.**—Recognizing the fatigue element in the production of writers' cramp, rest becomes the principal mode of treatment. All physical failings and conditions which tend to make fatigue of ready



appearance must be scrupulously sought and carefully managed. Absolute cessation of writing is of the first importance. Unfortunately, patients whose living depends upon clerical work will not or cannot conform to these requirements until the neurosis completely disables them. Exercises to develop the strength of the arms, hands, and fingers should be employed, and massage, baths, and electricity, for their general tonic effect, are of some service. Gordon claims to have secured immediate and lasting improvement by the local application of



FIG. 245.—1, Figure 1 to immobilize hand and wrist in the treatment of writer's cramp; 2, splint to brace.

Bier's method. When writing is again taken up, it must be by the proper full-arm movement. A typewriting machine can often be substituted for the pen. The use of special sedatives to control the cramp and permit a continuance of writing is pernicious, and rarely gives even temporary help. When writing is resumed, the free-hand method from the shoulder must be employed, and the splint (Fig. 245) affords a help by immobilizing the wrist and fingers during writing exercises.

#### OTHER OCCUPATION NEUROSES.

The general considerations pertaining to writers' cramp are equally applicable to the other occupation neuroses, and with proper modification the particular features are identical. Among the occupation spasms more commonly encountered are the cramps of violin and pianoforte players, telegraphers' cramp, seamstresses' cramp, and hammer cramp in smiths and artisans using the hammer. Artists, flower-makers, turners, watchmakers, knitters, engravers, masons in using the trowel, sailors from pulling on ropes, treadlers, compositors, cannisters, cigarette-makers, shoemakers, milkers, money-counters, letter-pressers, and players on various musical instruments, including drummers, comprise the list given by Gowers. It has been noted in a shoe salesman from the stooping position needed in putting on shoes, in gun-chewers, affecting the masticatory muscles, and in various factory employees who incessantly use the same movement in feeding or attending some machine. Clergymen and other public speakers, from a faulty use of the vocal apparatus, may acquire a laryngeal neurosis that is manifested every time the voice is strained, causing a sudden loss of modulation, which may end in continuous vocal disability. A spasm of a similar nature, involving the lumbar muscles, has been seen in physicians, due to continuous riding in cabs or huggies.

## CHAPTER VII.

## NEURASTHENIA.

IN 1869 Beard, of New York, directed general attention to a nervous state or condition marked by irritative weakness, and adopted for it the name of neurasthenia. It is familiarly known as nervous prostration or nervous exhaustion, and is a fatigue neurosis. In Europe it was at first somewhat derisively called the American disease or Beard's disease. It is now recognized the world over as a morbid state; as one of all time, and not a product of modern life or of American conditions. It is marked by a host of subjective symptoms and a very few objective phenomena, all more or less variable, and most of them inconstant. As a rule, all forms of nervous energy—psychic, motor, and organic—are reduced, so that fatigue is more quickly occasioned than in health. There is less endurance, and consequently greater irritability, which most shows itself in the mental sphere. The whole state is summed up in the classical term "irritative weakness."

**Etiology.**—Neurasthenia seldom occurs before twenty years of age or after fifty. It is essentially a disorder of the energetic, productive, troubled period of life. Both sexes suffer, and perhaps in equal proportion, but in females a more common association with hysteria frequently carries these cases into that list. Cleghorn,<sup>1</sup> basing his conclusions on statistics embracing 9000 cases, finds neurasthenia much more frequent in men than in women, and two-thirds develop between the ages of twenty and forty. Indose occupations bulk heavily in the list. In this country the high altitudes of the western plateaus and the extremes of climatic conditions in the northern, and particularly the northwestern, States apparently account for the greater frequency of neurasthenia in these localities than on the seaboard and in the southern States. The persistent winds of the prairie States seem also to be active in the causation of neurasthenia. All races present the disorder. Hebrews and Slavs are said to be very subject to it, and Scandinavians, at least in this country, furnish a large contingent. A *neurasthenic heredity* is less common than in hysteria or epilepsy, but neurasthenics are very commonly found at the head of a neurotic strain. *Debilitating conditions* in the antecedents of neurasthenics are very common. Gout, rheumatism, tuberculosis, syphilis, excesses, dissipation, malaria, and all the cachexias in parents are likely to disquiet the stamina of offspring and favor the early limitation of endurance and vigor demonstrated in neurasthenia. A defective education that omits discipline and the cultivation of self-control, poorly fitting the child for the rude shocks of later life, may be a predisposing cause. Educational methods that overtrain and overstrain may directly induce the neurasthenia. Occupations of all varieties furnish neurasthenics. It is only requisite that the element of overwork come in, whether the labor be mental or physical. Overwork, moreover, is a variable quantity, relative to the forces and endowments of the individual.

<sup>1</sup> "Med. Rec." April 27, 1907.



Among the *inviting causes* overwork is the most common, and associated with it we usually encounter anxiety, worry, or excitement, which depress the patient's forces, often at the same time impelling him to greater efforts. The business man, anxious for his ventures, works doubly hard to secure success. The sleepless mother, worn with care and nursing, does double and treble duty, and finally "goes to pieces" when the strain is over. The overtrained athlete goes "stale." *Excesses*, by their debilitating effects, are frequent sources of neurasthenia. Alcohol, tobacco, venery, masturbation, either as onanism or withdrawal, unnatural stimulation of sexual responses, and sexual continence after habituation may result in the general depression we call neurasthenia. *Trauma*, both physical and mental, may induce neurasthenia, and is likely to do so in proportion as the psychic shock is well developed. Railway accidents may breed neurasthenia in those who do not receive a scratch. The fright is often worse than the blow. Together they may cause a double injury. A muscular strain from overexertion, as in lifting, may start neurasthenia, particularly if the back be hurt and there is suggestion or fear of serious harm having been done. The more neurasthenia is studied, the more prominent will its mental side become. Exhausting illness, either from acute or chronic disease, may cause neurasthenia. Various *toxic states*, such as lithemia and syphilis, are prone to produce it. It is usually difficult, and often impossible, to determine the exact cause of neurasthenia in a given case. Ordinarily, there are a number of both predisposing and exciting causes. In a large number of instances neurasthenia is secondary to and symptomatic of organic conditions, such as phthisis, Bright's disease, diabetes, gout, rheumatism, uræmic and toxic states generally.

**Pathology.**—Although we know no pathological anatomy of the disorder, its manifestations are those best explained by a diminished dynamic energy and lessened recuperative power in the cerebrospinal axis, and especially in its cellular elements. This may be a nutritional defect. The very constant factor of persistent overwork, overstrain, and overfatigue in the causation of neurasthenia, coupled with Hodge's findings in the motor cells after an expenditure of energy, leads to the opinion that we have to do with a fatigue neurosis, general in distribution and comparatively slight in degree. With this view in mind, we will be the better able to understand the symptomatology and the requirements for treatment.

**Symptoms.**—The symptoms of neurasthenia are extremely numerous. Some of these are *essential*, most are *accessory*. Chiefly considered *kondashi*, *borbache*, *gastro-intestinal atony*, *neuramascular weakness*, *cerebral depression*, *neral irritability*, and *insomnia* as the fundamental symptoms of the disorder,—the true *stigmata* of the neurosis. Secondarily and momentarily arise a host of complaints that are of less importance and significance. It will be necessary to take up the symptoms seriatim. It may be said of them all that they indicate a deficiency of function, never an absolute want of it. The reduction is one of quantity rather than of quality, but is never absolute in degree.

**Motor Disorders.**—A constant condition in neurasthenia is *muscular*

weakness. The patients complain that muscular efforts are promptly fatiguing. They can only walk a few squares, standing a long time is exhausting, and exercise or use of the back and upper extremities prostrates them. They often show a remarkable diminution of strength as registered on the hand-dynamometer or in lifting, but occasionally a patient can put forth one or two fairly forcible efforts, and then the strength quickly subsides. Sustained effort is impossible. As another manifestation of the generalized myasthenia, *tremor* is frequently observed, and can usually be provoked by comparatively slight muscular efforts. Luzzati<sup>3</sup> found it in eighty-five per cent. of neurasthenics. Many patients complain of their trembling knees and shaking hands, or the tremor may appear in the handwriting, especially toward the end of a long letter, where the firmness of the strokes is also likely to be reduced. Tremor in the eyelids, lips, and face is sometimes noticed. Muscular twitchings in the face and extremities are not rare. The *tendon reflexes*, ordinarily, are increased. This is customarily the case with the knee-jerk. A tendency to widely distributed responses is frequently present, so that a tap on the patellar tendon causes starting of both lower limbs or of all four extremities, and sometimes gives rise to a complaint of pain in the back. If the knee-jerk be repeatedly produced, the intensified response first elicited may gradually subside, giving another evidence of the early fatigue of the nervous apparatus. In some instances a tap on muscle or nerve-trunk will call forth a similar response. Ankle-clonus is sometimes present, but is spurious in character, and only a few vibrations of the foot can be elicited. Repetitions of the test may fail to produce it. Paralysis or abolished knee-jerks are not found in neurasthenia unless due to other coincidental disease.

**Sensory Disturbances.**—Neurasthenia never causes *anesthesia*. When this is present we have a condition of actual deficit, to which limit neurasthenia does not go. The vague subjective disturbances of sensation, on the other hand, are limitless. Among them a feeling of general *hiredness* and *fatigue* is almost constant, and the recumbent posture tends to become habitual.

**Headache** is one of the most common symptoms; it is practically never wanting and often is described in striking terms. In some instances it is slight and constant, more often it is produced by any muscular or mental effort, and occasioned by any disturbing emotion. Usually it is occipital, "at the base of the brain," as these patients are fond of saying; but it may be frontal, temporal, or vertical. One describes a sensation as if the head were splitting or the skull lifting; another has a terrible weight or a severe constriction about the head. This *half-cep headache* is very common. Heaviness, throbbing, buzzing, a sensation of wind blowing or of water running under the scalp, and many others are perhaps indicative of the vasomotor disturbances within the skull. Almost never is the headache a veritable pain, but rather some variety of discomfort difficult to describe. *Backache* is equally common with headache. It is probably a fatigue symptom for the most part, though sometimes referable to gastrointestinal dis-

<sup>3</sup> *Berlin Neurol.*, August 15, 1897.



turbances. The small of the back is its usual seat, whence it streaks up between the shoulders or through the loins and down the limbs. Very commonly it is associated with a "drawing" sensation at the back of the neck, and the occipital pain is then usually complained of. By lying down or by padding a chair with pillows, some relief is obtained. The advanced case customarily assumes one of these expedients. In milder cases the backache is occasioned by any effort or disturbing circumstances and subsides upon rest.

*Tenderness* is usually found over the spine and is rather superficial in character, but occasionally is intensified by deep pressure. Rarely the spine is tender its entire length; usually only small sensitive spots are present. The upper cervical spine near the occiput, over the vertebra prominens, opposite the lower angle of the scapula, at the waist-line, at the top of the sacrum, and over the coccyx are the favorite locations. Sometimes the tenderness is diffuse and the skin over the entire dorsum is painfully over-sensitive. Sometimes this sensitiveness prevents the dorsal decubitus or interferes with sitting up. It is likely to be aggravated by anything which disturbs the patient or increases the other symptoms. Sensitiveness on the head, limbs, or other portions of the trunk is not rare, and is usually associated with *spontaneous pain* in the same location. The outlines of these sensitive areas are never sharply defined, and they may shift position or vary in intensity within a few hours or days, but sometimes remain practically stationary for months.

All manner of vague sensations of heat, cold, prickling, tightness, numbness, stiffness, weakness, fatigue, soreness, pain, pressure, etc., referred to this or that part of the body or limbs are constantly encountered. In addition there are a host of abnormal feelings referred to the thoracic, abdominal, pelvic, and genitive organs.

*Visual Disturbances.*—The neurasthenic commonly complains that reading has grown difficult because it causes headache and vague distress, and states that the letters blur or run together after a few minutes. In other instances they can not maintain attention. Careful examination will usually demonstrate that the *accommodative apparatus* and the *retinal sensitiveness* are promptly fatigued. One is a motor loss, the other a sensory deficiency. The first is shown by the rapidly diminishing ability to clearly make out the test-type; the second, by the quickly narrowing visual field, due to impaired sensitiveness in the retinal periphery. Both may be regained after a slight rest. A high and oscillating degree of muscular asthenopia is usually due to neurasthenia, and commonly subsides as the nervous state improves. Deficiency of the innervus is the usual finding.

*Photophobia* may be encountered of sufficient intensity to keep patients in dark rooms or wearing colored glasses. In lessened degree retinal hypersensitiveness is not uncommon, and is similar to the cutaneous sensitiveness. Misty obscurities and a veiling of vision are sometimes described, or everything looks strange and unreal. The pupils are usually very mobile, contracting and expanding excessively, sometimes sluggishly, sometimes very actively and even independently of light or

accommodative efforts. Transient inequalities are rarely encountered. Permanent inequality is due to organic disease.

**Disorders of Hearing, Smell, and Taste.**—Hearing is apparently frequently disordered. Thus, patients start at the slightest sound, and often tie up the door-bell and seek seclusion to avoid all such irritation. This is as much mental as aural, perhaps, but tinnitus in various forms and throbbings in the ears are due to the irritable weakness that spares no nerve. Complaints of peculiar or bad smells and tastes have a similar significance.

**Gastro-intestinal Disorders.**—Nervous indigestion is one of the commonest features of neurasthenia. The appetite is often capricious, and may be excessive or greatly diminished. The mere thought of food may be repugnant. In milder cases there are complaints of the food lying heavy or of gaseous eructations, which may or may not be attended by heart-burn. The gaseous gastric distention may suggest dilatation, and often provokes cardiac palpitation and precordial and epigastric distress. In more aggravated cases dilatation actually occurs, and hydrochloric acid may disappear from the gastric secretion. Digestion is retarded and deficient, but the tongue may remain clean and nutrition still be inadequate to maintain the body-weight. The *caecum* *intestinum* is usually affected in the same atonic fashion, causing constipation. In the severe cases of neurasthenia all these conditions are aggravated. The eructations of gas become frequent, noisy, and distressing; meteorismus, colicky pains, alternating constipation and anæmic diarrhoea, intestinal fermentation, and the passage of undigested food mark the atonic and irritable state of the gastro-intestinal tract. Cases present great variations, and the same case is rarely consistent in regard to these features.

**Circulatory Disorders.**—*Cardiac palpitation* due to digestive disturbance is one of the common symptoms in neurasthenia, but may result from other causes, as muscular or mental efforts, sudden starts or embroilments of any sort. In some instances it reaches a high degree and is attended by precordial pain, a tumultuous heart's action, throbbing arteries, and generalized distress. In other cases it may occasion a pseudo-angina pectoris that mimics a stenocardial attack very closely. A small, rapid *pulse* of about 90 a minute is commonly present in neurasthenia, and in attacks of palpitation it may reach 140 or 160. The rate is promptly increased in all cases by any distressing, exciting, or disturbing cause. Hemie murmurs are not rare in advanced cases, when the general nutrition has been reduced. The *feebleness of the circulation* is shown by the cold extremities commonly encountered. Vasomotor storms in neurasthenia are the rule. Localized or general flushings, sweats, and vertic throbbing may greatly annoy the patients.

**Secretory Disorders.**—The *urine* is ordinarily scant and high-colored. Neurasthenics drink little water, and the irritating urine may cause much vesical uneasiness and frequent micturition. There is commonly an increase of uric acid and urates, or a great abundance of phosphates and oxalates may be noted, and the urine may be neutral or alkaline in reaction. All *fluid secretions*, as the perspiration, saliva, gastric, intestinal, and synovial fluids may be deficient. In other in-



stances they are increased, or increase; and deficiency may alternate. Some patients complain that the slightest effort or embarrassment causes them to sweat profusely, usually about the head and neck. The cold, clammy hand of neurasthenia is rarely wanting.

**Genital Disorders.**—The average male neurasthenic complains of *lessened sexual power*. If unmarried, nocturnal emissions frighten him, and he is convinced that he has spermatorrhea by the cloudiness of his urine. If, perchance, there has been an antecedent habit of masturbation, every symptom is warped by him into relation with the supposed impotence, and he develops into a confirmed sexual neurasthenic without erections and with vague parasthetic sensations or actual tenderness and pains in the genitals that claim his undivided, brooding attention. If married, premature ejaculations and loss of sexual appetite in milder cases are followed by actual inability in the severer forms, and some sensory hyperæsthesia or lessened sensitiveness of the parts may be found.

In women analogous eruptions are encountered, but much less frequently. Some are troubled by nocturnal orgasms accompanying dreams from which they awake nervous, depressed, and exhausted. In married women sexual appetite may at first be somewhat increased, but quickly diminishes and commonly disappears, not seldom being followed by an actual distaste or even disgust. Pelvic pains and genital insensitiveness, or hyperæsthesia, are often added, and further serve to inhibit intercourse.

**Mental Disturbances.**—The psychic side of neurasthenia is an interesting and important feature of the neurosis. Indeed, the condition may well be denominated a *psycho-neurosis*. All the mental manifestations of the neurasthenic have in common the same elements which dominate the physical features—namely, weakness and irritability. The capacity for mental work is *abridged*, just as the muscular power is reduced. Prolonged mental efforts become irksome or impossible, and in many instances attempts to read or think or converse for a few minutes produce so much fatigue and discomfort that they must be discontinued. This mental *asthenia* also shows itself in the reduction of the mental concentration necessary for fixing and maintaining attention. Neurasthenics can not keep their minds on the printed page nor follow a line of thought for any great length of time. They are inattentive to details, and consequently do not clearly apprehend them. Hence arises the almost constant complaint of a *loss of memory*. Active *spontaneous sensations*, which is dependent upon mental strength, is also reduced. Ideas do not occur to such patients with their usual vigor and rapidity, and they often assert that they can not think. Their *consciousness* naturally subsides with their strength. Attracted by the cardiac palpitation or their sexual incapacity or their mental debility, or by any other neurasthenic index, they become *introspective*, and, misconstruing their symptoms, develop a lot of *complexions*. As the suggestions and reminders of these fears are constantly present, the neurasthenic becomes saturated with them, thus still further reducing the power of attention and memory. They become morbidly self-watchful and tend to hypochondriacal depression. In some instances these morbid fears pass into *mania delusions* and obsessions, and are then beyond the boundaries of neuro-

thinks, but the dividing line is decidedly indefinite. Indeed, many psychoses begin as a neurosthénia and even general paresis may show a neurosthénic prodromal period.

*Fear*, arising easily from a consciousness of weakness and a loss of courage, often becomes associated with external conditions and suggestions. An attack of palpitation in a crowded place may induce a coalition of fear or apprehension constantly associated with like conditions. Some fear to be alone, others to be in open or in narrow or in high places. Some fear darkness, some storms, some lightning, some special localities, some contamination by dirt or infection by disease. The neurosthénic always recognizes the baselessness of such fears, though he may not be able to dispel them; the insane hypochondriac accepts them as actual necessary facts, and can not be persuaded to the contrary. When in neurosthénia the phobias become dominant, so that the patient is controlled by them, though recognizing their fictitious character, the condition constitutes an actual *psychasthénia* in the sense employed in the next chapter.

Frequently the neurosthénic recognizes a *diminished effort* for his family or others, and sometimes is greatly troubled by it. He is irritable, peevish, fault-finding, and resentful. The business man dreads his daily tasks and dislikes to meet new customers or even old friends. The *confidant* is less stable, and there is ever a tendency to weakness and depression. Such patients are lachrymose on slight occasion or break down in telling of themselves or in contemplating their darkened future.

*Sleep* is commonly faulty from the first. Neurosthénics habitually present *insomnie*. In some instances there is a difficulty in getting to sleep or the sleep is constantly disturbed. Exceptionally the sleep is sound and prolonged, but the patient awakes *unrefreshed* and usually more depressed and prostrate than upon retiring. Ordinarily, the sleep is disturbed by troubled *dreams*, which often are of a dreadful or night-mare-like character. In shock or traumatic cases the accident episode may be repeated in the dreams night after night or several times the same night. Charcot laid especial stress upon such formulated dreaming in neurosthénia.

**The General State.**—In neurosthénia of long standing the general nutrition is commonly defective, and high degrees of *anæmia* and *emaciation* may be encountered. Neurosthénia of a moderate grade of severity, but sufficient to disable the patient for ordinary pursuits, is consistent with fair nutrition, and some of the most inveterate cases may become quite fat. Emaciation, on the one hand, is not a hopeless indication, nor, on the other hand, does plumpness indicate an easily manageable case.

**Forms.**—In view of the multiplicity of symptoms in neurosthénia and their lack of uniformity in various patients, it is not surprising that numerous forms have been described. The terms *cerebral*, *spinal*, and *sexual neurosthénia* have been used to designate cases in which symptoms referable respectively to the head, back, and genitals have preponderated, but it is best to look upon neurosthénia as one and indivisible. Adventitious or exaggerated symptoms may variously tone the clinical picture in different cases, but neurosthénia remains a generalized disorder



of the entire nerve-apparatus. In the foregoing description of neurasthenia the simple variety has been constantly in view. We are always to have in mind that neurasthenia may be *symptomatic* of some organic process of which it is a secondary expression. Thus, it may follow all forms of extraordinary illness or injury, spring from the various exotoxins, and succeed all toxic and infectious processes. All such conditions must be eliminated before we may call a case one of *simple* or *essential* neurasthenia. Again, neurasthenia is frequently encountered in *association* with other neuroses. This is particularly true of hysteria, and it is sometimes impossible to say where the neurasthenia ceases and the hysteria begins. For such cases the term *hysteroneurosthenia* has been employed. *Psychasthenia* is frequently associated with neurasthenia.

**Course.**—Neurasthenia is essentially a *chronic* *malady*. Ordinarily, its onset is insidious, and it is only after weeks and months of growing incompetency and laborious efforts to carry on the usual duties that the patient recognizes something wrong and seeks medical advice. In the traumatic form of neurasthenia the neurosis may be quickly established, but it is not uncommon for the patient to go about as usual for several days or weeks after the accident or injury, and gradually develop the nervousness, weakness, and prostration. Once established, neurasthenia tends to persist indefinitely, and only some radical change in the mode of life, serving to relieve the patient of worry and work, is likely to favorably modify the condition. Frequently remissions are presented, but the patient relapses under any unusual demands, and slowly or partially regains the former status. Even after long periods of improvement there is a tendency to ready recurrence under the influence of any exciting cause.

**Diagnosis.**—The diagnosis of neurasthenia is usually easy. The combination of mental and physical weakness and irritability with gastrointestinal atony, tachycardia, headache, insomnia, and rapidly exhausted retinal sensitiveness, justifies the diagnosis. The difficulty mainly lies in overlooking some basic organic disease. One should never be content with a diagnosis of neurasthenia until he can thoroughly satisfy himself that he is not confronting a symptomatic form of the neurosis. *Hysteria* may be associated with neurasthenia, but has its own stigma. It is to be kept in mind that disturbances of function in neurasthenia are those of irritable weakness and not of actual loss. Anesthesia, paresthesia, convulsions, complete loss of self-control, actual amnesia, are not the property of neurasthenia, but are the out-marks of hysteria. True *hysteroneurosthenia* is a variety of melancholia with actual insane delusions regarding bodily states. In neurasthenia the phobias are under at least partial control, and sometimes can be completely relieved by a reasonable statement of the facts. As before stated, all the changes of function in neurasthenia are in the nature of quantitative reduction.

**Prognosis.**—The outlook in neurasthenia is commonly good if the proper treatment can be instituted and maintained. It is often very difficult to secure the cooperation of the patient, or his circumstances may be such as to preclude the protracted rest and change usually required to restore him. In that event he suffers along as best he can, possibly picking up some drug or stimulant habit, and may recover after many

months, but is more likely to break down completely and become a confirmed nervous invalid. If of a psychopathic stock, some pronounced mental change may appear. Cases occurring before twenty years of age are likely to yield readily and to relapse frequently. Such early demonstration of insufficient stamina naturally indicates an unstable organization. After forty the prognosis is also less hopeful, as the neurasthenic exhaustion occurs in tissues that have largely lost their resiliency and recuperative powers. After this age full vigor is practically never regained if the neurasthenic state has been severe. A neurotic or psychopathic heredity or evidence of degeneracy in the patient reduces the chances for a full and permanent recovery. The bodily condition is of less significance if organic disease is absent. Cases of extreme emaciation often respond admirably to treatment, while some of the cases without marked disturbance of bodily function are inveterate.

**Treatment.**—The keynote of treatment in neurasthenia is *rest*. Depending upon the severity of the case, upon the temperamental characteristics of the patient, and upon the sex, this rest may be partial or as complete as we can possibly make it. In the milder cases, especially those occurring from overwork in sedentary pursuits, in patients between the ages of twenty and forty, and when the patient's make-up is based upon a good heredity, the full Wier Mitchell rest treatment is commonly not required. It may only be necessary to have the patients spend four or six additional hours in bed daily. This can be secured by having them retire shortly after the evening meal, and not rise until late in the morning. A hot bath of five minutes' duration and a glass of hot milk or a glass of beer on retiring for the night encourage sleep. A cold douche or cold sponge, to the full length of the spine on rising, gives a vigorous morning start. In addition, an hour's rest in the recumbent posture in the middle of the day should be enjoined. The amount of work must be reduced. It is rare that this can not be done even in the most responsible positions. It should be needless to say that the condition of *organic activity*, if deranged, must be put to rights. Constipation, a sluggish skin, or inactive kidneys, if uncontrolled, will defeat the best general management. Large quantities of drinking water usually aid in several ways, and, as a rule, neurasthenics shun water. If these regulations can be instituted during a vacation, especially one spent among nice scenes, and sometimes with light and interesting occupation, an additional advantage is gained.

In the *severe male cases* an absolute separation from business and family is usually required, and a long sea voyage with a pleasant companion often works wonders. As a rule, the more outdoor air and recreation that does not entail effort, the better, but to put a neurasthenic on a bicycle or on long walks adds fuel to the flames. A carefully selected, *nonstimulating, soothing, nourishing diet* is of considerable importance. If the scales show a gain of a few pounds, success is assured. Men, unless practically bedridden, do not respond favorably to the Mitchell rest-treatment. The enforced illness and confinement to bed is rarely tolerated by them if they can possibly be absent.

In the *severe female cases*, and sometimes in young males or completely prostrated men, we must have recourse to the full rest plan laid



down by Mitchell. The details of this are furnished in all modern works on therapeutics and need not be repeated here. By this plan expenditure of energy is reduced to a minimum, strength is conserved and increased by the massage and electricity, and flesh is rapidly made by the constant feeding. There are several essentials in carrying out this procedure, and their neglect means failure. The first is isolation from relatives and friends, and the establishment of an entirely new and hopeful atmosphere. In some instances the slightest infraction of this rule causes immediate relapse. Equally important is it to have an experienced and tactful nurse.

Any amount of general hospital training does not make a good nurse for this class of patients or furnish the endless tact and self-repression needed to manage them. It is difficult to induce nurses, accustomed to grave operative cases and severe physical illness, to appreciate that the endless complaints and fault-findings of neurasthenic patients are symptomatic of their state and not merely petulant crankiness. It is also very important that the treatment take place in a suitable location, free from disturbing noises. The room and immediate surroundings of the patient must be bright and cheerful. If isolation with a suitable nurse, preferably one able to give massage, can be had, the other details of treatment are less important. The absolute milk diet does well in some cases, and is indispensable in a few at the beginning, but if food is digested and the bowels active it is immaterial what the patient eats so long as it is nourishing, abundant, and administered at frequent intervals. Sleep, usually disturbed, under the massage and forced feeding soon becomes sound and prolonged. Hot milk, an alcohol rub, or a glass of beer at bedtime is commonly a sufficient hypnotic.

No scheme of treatment in neurasthenia is complete that ignores the mental element of the disorder. These patients are frequently impressionable, and particularly so as regards their own health and prospects. Their fears can not be laughed to scorn. They should be carefully estimated, fully explained, and then dismissed. It is not advisable to allow these patients to reiterate their complaints to the physician and the nurse, and the family must be instructed to refuse to hear or discuss them with the patient. Encouraging suggestions and hopeful assurances constantly repeated have the force of hypnotic suggestion, and a very real therapeutic value. For this reason the physician must usually see his patient frequently. In this way the daily use of static electricity, hypodermic injections of strychnin or water, or any other objective excuse, impresses the patient and permits and emphasizes the repeated encouraging suggestions. With all the physical measures is to be continued a re-educational psychotherapy. Mental and physical tasks, activities, and work must be adjusted to the patient's abilities and capacities and this training must go forward with him for months and sometimes for years.

Thus far nothing has been said about drugs, and there is very little to say. Iron and arsenic against the anemia, trional and bromal for the insomnia, laxatives for constipation, may be given. Strychnin in large doses, or bromids and sedatives in large doses, are ill-advised and

commonly harmful. The indications are to rest and to nourish, not to stimulate or to depress. *Hydrotherapeutics*, especially if carried on at a distance from home, yield good results in mild cases. It may be said that the treatment of neurasthenia requires judgment, tact, perseverance, and personal force of the highest order. When the neurosis is secondary to organic processes, its treatment is similarly second in order. *Climatic* conditions embracing windy low-lying stretches, altitudes above 3000 feet, and fluctuating extremes of temperature are decidedly disadvantageous. As a rule, neurasthenics do best at the sea-level and preferably at the seaside in an equable climate.

## CHAPTER VIII.

### PSYCHASTHENIA.

OCCUPYING a middle ground between nervous diseases and out-spoken insanity, sometimes linking them together, are a large number of manifestations of reduced mental control or of limited mental weakness to which the term psychasthenia may be well applied. Such patients usually seek relief of the general practitioner or the neurologist and are generally considered as nervous or neurasthenic, hence the propriety of discussing them here. While their mental manifestations are analogous to those of neurasthenia, hysteria and actual mental alienation, in typical cases and in large numbers they furnish a fairly definite syndrome devoid of the irritable weakness and ready fatigue of neurasthenia, the stigmata of hysteria and the erroneous ideas of insanity. Naturally occurring in neurotic and degenerate individuals, it would be strange did they not show a mingling with or a development into the other psychoses and psychoses. It is even stranger that such bizarre mental activity as is shown by psychasthenics may be of lifelong duration without essential variations and without forcing the patient into the field of insanity.

Under the term psychasthenia may be grouped those conditions which have been variously termed fixed ideas, obsessions, imperative conceptions, impulses, morbid scruples, phobias, doubts, agitations, feelings of strangeness, feelings of changed personality, anxiety neuroses, mental and motor tics, etc. In all there is a certain enfeeblement of psychological functions of protracted duration, and in all a clear recognition by the patient of the unreasonable nature of the act, idea, or condition, and of the defective self-control.

The fixed ideas, obsessions and phobias pertain to nearly every subject of human feeling and activity, and are all attended by a sense of dissatisfaction, but rarely by great mental depression. We may, with Janet,<sup>1</sup> classify the obsessions as those of sacrilege; of crime; of shame of self or person; and those of a hypochondriacal sort. For the phobias the Greek language has been searched to find sufficient names. Claustrophobia, fear of closed places; agoraphobia, fear of open places; amaxophobia,

<sup>1</sup> "Les Obsessions," Paris, 1901.



fear of vehicles; misophobias, fear of dirt; and a hundred others testify to the range of human feeling and the wealth of the Hellenic tongue. In many cases, however, the fear is vague and indefinable. Janet has laboriously tabulated the phobias as: fears regarding the body; fears of objects; fears of situations, physical and social; and fears of certain ideas. Under these heads he has listed over forty different varieties with innumerable variations.

These mental symptoms, while often recurring for many years in the same patient, are not continuous. Like the motor ties (see p. 614), which are merely the enactment of fixed ideas, they are intermittent, but of more or less frequent occurrence. Again, like the motor ties, those well called mental ties are prone to remain precisely the same for a given case, or when modified the change is either an evolution along functional and associated lines or a reduction to a part of the original mental concept. These fears, impulses, etc., therefore, occur in attacks, crises and bouts with varying intervals of comparative calm.

The origin of mental ties usually has relation to some emotional shock or striking experience, but their repetition may be determined by any object, circumstance, sound, smell or other suggestive factor, however remote. Finally, they may occur with almost spontaneous rhythm, like the running of a tune through the head. A locality where once a patient has experienced the mental anguish of an attack of claustrophobia is indelibly associated with that episode and is usually shunned to prevent a recurrence. Such patients in a similar way may continuously narrow their opportunities and shorten their radius of action until they may finally confine themselves to the limits of the home or even to those of a single room. In the dread of contracting or transmitting a contagion some refuse to shake hands and finally to even touch articles or garments used or to be used by others, and all the time with the angry expression that they know it is all nonsense but that they cannot help it. In fear of poison they may prepare their own food or only partake of that first shared by others. Those impelled to crime take endless and often perfectly silly and inadequate but conflicting precautions to prevent the act which all the time they declare they contemplate, yet feel they will never accomplish, and as a matter of fact these morbid impulses never culminate in any serious crime. The whole story is the recurrence of the idea. Patients who are morbidly impelled to touch certain objects, to take steps in a certain manner, to turn in an exact fashion, to repeat words or lines when reading, to retrace a certain number of steps, do these things habitually and are made extremely uncomfortable, almost frenzied in extreme cases, if prevented. If any of their manoeuvres are foregone they describe feelings of unrest, distress, vague apprehension, and may be made very unhappy thereby, but will readily agree that there is no sense in their acts, fears, or worries, yet insist that they cannot control them. As a matter of common observation, however, they at times can and do control themselves. In all its phases, therefore, this mental condition is one of incompleteness. There is about it a certain laughable incongruity quite apparent to the patient, who may only tell of it in a joking, shamfaced fashion.

In the crisis of obsessional attacks the patient may clearly manifest his perturbation of mind and its physical effect. Anguish and fear are shown in the countenance and in the attitude and actions. A dilated pupil, a pale face covered with perspiration, rapid pulse and respiration, are the physical concomitants of the mental agitation, but the patient does not lose consciousness, though the mental storm seems irresistible, and he experiences a feeling of relief on its termination.

In the **etiology** of psychasthenia heredity plays an important part. Pitres and Regis in the tabulation of 100 cases could find but 20 in which there was no prenatal trace of nervous or mental instability; Janet in only 8 out of 100. The great majority of psychasthenic individuals are clearly marked by the signs of degeneracy. As to sex, females preponderate about 3 to 1. While no age is exempt, the most favorable period for the development of psychasthenia is between twenty and fifty, with the maximum at about thirty.

The determining causes are almost innumerable, but importance attaches to fatigues, excesses, traumatism, infectious diseases and developmental and evolutionary life periods. Gastric and cardiac disturbances, genital diseases and conditions, vertiges and other marked sources of self-concern bulk rather largely in the list. Freud<sup>1</sup> is disposed to regard the sexual element as pre-eminent if not invariably in operation in the causation of psychasthenia as well as in hysteria. Psycho-analysis by the Freudians frequently uncovers some peculiar sexual experience which the patient may then be persuaded is the cause of his obsession and sometimes with complete relief, but there are other than sexual causes of psychasthenia. As emotional determinants we may enumerate the influence of strong religious emotions, the self-questionings of adolescence and sex relations, frights of all kinds, death in all forms, fear of all diseases, grief, bereavement, and deprivations. In a general way emotional states long continued are more potential than emotional shocks.

The **course** of psychasthenia is essentially chronic and we cannot with propriety place a case in this catalogue that has not persisted for several months. The onset proves to be insidious the more carefully we analyze the mental features that precede its symptomatic appearance; in only a very few cases does it appear to come suddenly in the immediate wake of an emotional shock, and even here predisposing features are commonly well marked. A period of self-questioning, of mental rumination, of premeditation is commonly crystallized into the particular obsession that thereafter has a partial and sometimes almost complete domination of the individual and which may last for the rest of life. Many psychasthenics regain their self-mastery almost completely; to relapse on some further provocative occasion, the condition being remittent, or it may actually intermit and recur in a modified form. Occasionally one idea will replace another of a similar nature.

Physical states dependent upon such morbid ideas as defeat the proper manner of living—fear of food, of urination, of defecation, of sunlight—may powerfully modify the general health and furnish physical complications. Janet insists that psychasthenia being a psychological affair is not foreign to insanity, and out of 300 cases noted 23 who became definitely alienated

<sup>1</sup> "Wiener klin. Wochenschr." 1899.



mentally. The tendency shown by these cases was to the elaboration of fixed and systematized delusional states.

The termination of psychasthenia frequently brings the individual to a condition of inertia and self-isolation. Many gain a relative mastery over their obsessions. A few make an apparently good and lasting recovery. Remissions and relapses and intermissions are common. A small portion become absolutely insane.

The **diagnosis** in a typical case is perfectly plain, but the association of fixed ideas, phobias and obsessions with neurasthenia, hysteria, and insanity, makes of the first importance a careful scrutiny, to determine the presence or absence of these states. The features of good physical and relatively of good mental health, aside from the obsession, are important. The absence of somatic and stigmatic indications of hysteria and neurasthenia, and the fact that the patient fully recognizes the groundlessness of his morbid idea, are strongly indicative of the psychasthenic nature of the obsession. Any association of these diseases is of course possible.

The **prognosis** in psychasthenia is sufficiently indicated in the statement of its course. However ludicrous this psychoneurosis may appear in minor phases, it may readily incapacitate an individual for the usual walks of life and finally by its domination make a slave prisoner of him. Once recognized as an evidence of perenial limitations the physician is in a position to understand that he is dealing with a deep-seated, inherent defect, and that a long struggle is in view for him and his patient. Yet many cases are manageable and a practical recovery may be often secured.

The **treatment** of psychasthenia, aside from the prophylaxis, which embraces the control of parentage, etc., is physical and mental, but especially mental.

The physical treatment aims to discover and remedy any and every local or general deviation from health. It embraces the rules of hygiene and dietetics, measures of tonic and sedative medication, and directions for physical exercises.

The mental treatment of psychasthenia is a far-reaching subject. It first of all requires a physician sure of himself, sure of his diagnosis, and well equipped to understandingly enter into the patient's feelings and to assist him by various mental crutches to regain his self-control and master his obsession. The patient in a certain profound sense requires re-education and his life must be singlified. He must have a definite purposeful occupation. He must practice self-control, and his discouragements must be explained and his courage supplemented by the advice and support of the physician. Many such patients find great comfort if their condition can be attributed to some physical cause the remedy of which is fraught with hope. Others can be best managed by a full analytical discussion of their symptoms and conditions. Others can be aided by the frequent use of some placebo, the taking of which arouses and repeats in their minds the helpful suggestions of the physician and tends to the maintenance of a hopeful expectancy. Every time they control their fears or impulses they gain strength to master themselves, and every time they yield they lose correspondingly. Electricity in its multitudinous applications, hydrotherapy, massage, are useful, but mainly

as an avenue of suggestive therapeutics, and when thus applied with the constant assurances of benefit do much good.

Suggestion under hypnosis sometimes gives brilliant, but usually temporary, results. It cannot be made the sole reliance. Change of surroundings which have become charged with potential morbid suggestions and associations, the recreation of travel and the stimulation of objective interests, have great value. Everything must be done to change the self-centered, egotistic point of view.

For further matter relative to psychotherapy as applicable to this condition the reader is referred to the portion of this book dealing with mental diseases.

## CHAPTER IX.

### MOTOR TICS.

**Tic, Maladie des tics, Mimic Spasm, Habit Spasm.**—French writers, following Tourette, make a sharp distinction between a tic and a spasm. After them, a motor tic is an action which is identical with movements of volitional intent, and contains, therefore, a psychic element which may be subconscious. It is a psychoneurosis, a variety of psychasthenia, and is accorded a separate description mainly by reason of its objective motor features, which are often confused with other disorders. The preceding chapter should be read in order to gain a fair comprehension of its nature. In facial tic attention is called to the winking of the eye, exactly like that which excludes a flying particle of dirt; to the movements about the mouth and nose, identical with those produced by sensations of taste or smell, and to the occasional functional association with those of swallowing efforts, laryngeal motions, the production of sucking or smacking sounds, of grunting, and of articulate words, sometimes of an indecent character (coprolalia). A facial appearance that is expressive of some emotion, as of grief, pain, fright, or joy, may be repeated by the tic. Some cases, becoming more wide-spread, involve the neck and upper extremity so that attitudes and gestures are produced in conformity with the underlying mental idea. These tics, of which *lépharysme* is a type and *coprolalia* the extreme development, are more or less under the control of the patient, who can, by an effort of will, do considerable to repress them. After such repression "tictors" are likely to feel more or less vague discomfort and often yield to a regular spasmodic debauch, which seems to give them a feeling of relief. During sleep the tic completely subsides. Such patients frequently present a most marked neuritic tendency, and sometimes other neuroses, as writers' cramp and hysteria, or mental and moral obliquities are present.



Ordinarily, a tic is rapidly executed, and may be repeated with great rapidity from two to scores of times, when a lull occurs for a few minutes, or perhaps an hour of quietude may intervene. Any excitement or embarrassment promptly recalls and intensifies the morbid motions. On the other hand, any decided interest fixing the patient's attention interrupts the twitchings. The tic has a tendency to invade neighboring muscles of associated function, and frequently becomes bilateral, but is seldom symmetrical, excepting those tics more or less limited to the face, especially to the nose and mouth. While, ordinarily, the move-



Fig. 746.—Facial Tic.



Fig. 747.—Mental Tic.

ments are abrupt and momentary, they may, in a part or the whole of their distribution in long-standing cases, present to the features of greater or less duration. In some instances the eyelids are so firmly closed that the pressure upon the eyeball is painful. Rigidity in the lips is complained of at times as a feeling of stiffness, and the action of the zygomatics and buccinator may keep the angle of the mouth persistently retracted and elevated. The neck may be rigidly held in a given position. A somewhat similar spasm is often noted after Bell's palsy. It is, however, always unilateral, the affected muscles are distinctly contracted (see page 123), and it does not so distinctly imply purpose. However violent the motor tic may be, as Janet insists, it never results in any physical hurt to the patient or damage to his surroundings. Its inconvenience consists mainly in making him conspicuous and self-conscious. Although motor tics may imitate useful movements and gestures, when once established they serve no useful end whatever.

**Etiology.**—Youth is the preferred age for the development of tics, but adult life is not spared. An appreciation of the mental substratum of tics enables one to comprehend something of their genesis and in-

tractability. There is no doubt that many cases of blepharospasm originate in some irritation of the ocular apparatus that forces the attention of the patient into a groove leading to a mental and motor habit. These ties have for long been well called habit spasms. The term habit chorea, also applied to them, is misleading, though they may be grafted upon a chorea as a sequel by suggestion. In the same way a protracted grief, despair, or ecstatic pleasure may, in one neurotically predisposed, furnish the subconscious factor for an expressional tic, which reproduces exactly the faces of the underlying idea, perhaps made grotesque by its unilateral distribution. The thread can sometimes be followed by covering the sound side of the face and trying to interpret the emotion expressed by the tic. Sniffing, swallowing, and phonation are merely the manifestations of functional association, and regurgitation is but the vocalization of the imperative concept that may be otherwise subconscious. A facial spasm is sometimes associated with neuralgia of the fifth nerve, and bears the distinctive and classical name of *tic doulosoreux*; the spasm, however, is secondary to the pain and sometimes expressive of it. It is a grimace, but may persist as a true tic after pain ceases. A tic in the limited sense is not painful, though patients often complain of aches and tired feelings in the muscles affected by the prolonged contractions of postural ties, as, for instance, in mental torticollis.

**Varieties.**—Aside from the habit movements of idiocy and dementia, which constitute reversionary or degenerate ties, we may enumerate blepharospasm, facial spasm, spasmodic torticollis, mental torticollis, the latak of India, the myriachit of Siberia, the jumpers of New England and Canada.<sup>1</sup>

In mental torticollis<sup>2</sup> some deviation of the head is customary and is spasmodically maintained. Ordinarily, it ceases when the patient lies down or it can be controlled by a slight amount of manual pressure upon the head or face.

Jumpers and the subjects of latak and myriachit execute any one of several commands or suggestions impulsively, often violently, and frequently against their apparent will. Thus, upon command, they may strike, jump, or undress themselves.

**Treatment.**—In all cases of *facial tic*, after general measures, we are to look for and correct any peripheral irritation that can be associated with the seventh nerve. This is particularly the case in the distribution of the sensory portion of the fifth, but irritation arising even at a distance, as in the intestines or pelvis, may maintain the tic, and when corrected the tic may subside. Pressure upon certain points in the distribution of the fifth, first described by Græfe, often checks the tic. The most usual one is at the supraorbital notch. In a general way they correspond to the tender points of Valleix and the maxima of Head. We should always and repeatedly search for them carefully, going over the neck, shoulders, and upper chest, as they are sometimes distantly located. By exerting pressure upon such a part, the tic seems

<sup>1</sup> *Am. Coll. Surg. Med. News*, Dec. 11, 1895.

<sup>2</sup> *Hospiers*. "Thèse de Paris," 1893, and Brissaud, "Léçons," 1895.



to be reflexly inhibited, and the habit is arrested, at least temporarily. By repeating the arrest frequently and for long periods, the habit may be fully broken. In the same way the use of faradic electricity will occasionally render service. For this purpose the muscles engaged in the tic should be thrown into powerful tonic contractions for a number of minutes, from five to fifteen. One pole taken in the hand and the other placed over the stylomastoid foramen, or on the particular facial branch indicated, makes a suitable arrangement. The patient may advisably keep a battery at hand and use it as often as the tic becomes marked. It for the time being teaches the muscle a steady contraction and perhaps favorably affects the mental state by the peripherally induced suggestion. At any rate such application is often followed by temporary marked relief, and, in fortunate cases, by practical cure. A sugar pill, to be taken with great exactness every thirty minutes, has served a good purpose by constantly reminding the patient of the physician's suggestions, and recalling his attention to voluntary self-control.

Finally, nerve-stretching may be employed. If thoroughly done, it induces paralysis for a longer or shorter time, but, unfortunately, a restoration of motor function usually is marked by the reappearance of the tic, for which the irritation in the healing nerve-trunk may be responsible. A tendency to relapse in these cases is marked. The deeper the mental taint and the more pronounced the neurotic background, the less are they manageable. It is not impossible that suggestion, by reaching the subconscious element, may sometimes yield favorable results. All varieties of retention dressings and appliances are at first apparently helpful, but shortly become irksome, unbearable, and harmful.

In *mental torticollis*, Feindel<sup>1</sup> has reported good results by massage and exercises, the purpose of which was to build up the patient's self-control and mental stamina. With these he associated encouragement (suggestion) and general tonic measures. The exercise treatment embraces two varieties of motor effort. First, the patient practises voluntarily controlling the spasmodic muscles by keeping them at rest. Second, the affected muscles are alternately contracted and relaxed methodically a dozen times. These exercises should be repeated hourly. Similar exercises for the lesser facial tics and re-educational self-control measures are frequently successful. It is only by attacking the mental element, the obsession, that the psychoneurosis can be managed, and the various suggestion measures are a means to that end.

<sup>1</sup>"*Neur. Jour. de la Salpêtr.*," Dec., 1897; also Menge et Feindel, "*Les Tics et leur Traitement*," Paris, 1892.

## CHAPTER X.

## HYSTERIA.

HYSTERIA has been an interesting problem since the earliest days of medical thought. Unrecognized it occasioned the demoniacal "possessions" of the middle ages, and furnished some of the martyrs of witchcraft and religious fanaticism. Affecting whole communities, it caused epidemics, allayed by appeals to St. Gue, St. Vitus, or other tutelary. It has been seen in the excited religious gatherings of all countries. As a disease it was long supposed to be limited to the female sex, and was attributed to vapors or other influences arising from the womb; hence the name *hysteria*. Only in recent years have the endemic and epidemic forms been understood, and the male found to share with the female in the liability to this psychoneurosis. For many years the mental element in hysteria was at least partially recognized. Morebais used the definition, "A state in which ideas control the body and produce morbid changes in its functions." So far as the paralyses and contractures are concerned, English writers described them as "depending upon idea." The studies of Charcot and his students placed hysteria upon a rigid clinical basis, and traced all of its manifestations to disturbances in the psychic sphere or in its substrata.

Janet, who for years studied the psychology of hysteria, early contended that "Hysteria belonged to a group of mental diseases of cerebral insufficiency." In a more recent pronouncement he said: "Hysteria is a form of mental depression characterized by the retraction of the field of personal consciousness, and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality."<sup>1</sup> He attaches great importance to the rôle of "amnesia," or absent-mindedness, through which certain experiences drop out of the field of consciousness, with a resultant change of personality which eventually may become doubled or variously fragmented.

Babinski, who would materially limit the physical manifestations of hysteria, says: "Hysteria is a special psychical state capable of giving rise to certain disturbances, which can be reproduced by suggestion and removed by persuasion (suggestion)." On this basis he erects a rather arbitrary criterion beyond which he sees nothing hysterical.<sup>2</sup>

The very interesting views of Freud trace all manifestations of hysteria to mental processes, more or less morbid, in which motives of shame have resulted in repressing the consciousness of certain experiences, which thus become unconscious or subconscious. In this state they tend to symbolize themselves, and hysterical manifestations more or less locally or functionally related arise. He traces the cause of the repression invariably into the field of sexuality, which with him is so wide as to embrace everything bearing any relation to sex impulses and feelings. Shame, modesty, parental and filial affection, and all sexual experiences and perversions lie within this domain. The "shell shock" cases of re-

<sup>1</sup> "Major Symptoms of Hysteria," 1907.

<sup>2</sup> "Démembrement de l'Hystérie," etc., "La Sem. Méd.," 1909.



cent military experience would seem to sufficiently indicate that sexual traumata are not the only mental factors in the causation of hysteria. The practical part of Freud's psychology consists in the contention that the motive for repression being once discovered, the patient can re-establish a normal mental attitude toward the circumstance, and its train of hysterical symbols then disappears.

The purpose of citing these various views is to draw attention to the fact, in which they all agree, that hysteria is a mental disease, and that it must be so treated by the physician.

In addition, there are a number of organic phenomena—disturbances of nutrition, trophic and vasomotor disorders—of a neurotic character. Hence, we class hysteria as a psychoneurosis. The absence of detectable primary changes in the nervous system is admitted on all sides, even in very chronic cases.

In the presentation of the subject of hysteria the general outline of Charcot and his school will be followed. It is recognized that the type cases on which their descriptions were based are unusual, and even to some extent artificial, but they were complete. By understanding the full range of symptom groups, one is enabled to recognize less complex instances of a similar nature, and hysteria is essentially a disorder of many fractional parts, which may be encountered practically alone or in various combinations.

**Etiology.**—Hysteria in slight or severe form is one of the most common of nervous diseases. The age of puberty and the years of adolescence immediately following furnish the majority of cases. After twenty-five the frequency of hysteria declines and it becomes rare after forty-five. Before ten it is also uncommon, but children may develop it in very marked form even as early as two or three years of age. The sensory features so common in adult hysterics are rarely encountered in children. Formerly considered almost exclusively limited to the female sex, later statistics go to show that males and females are affected with hysteria in nearly equal ratio. According to Marie, in the lower social levels males predominate; in the wealthier classes females are more commonly affected. Hysteria is a disease of all countries and all races, but the Latin, Slav, and Israelite may be considered as particularly liable. Heredity plays an important part. Hysterics usually belong to neuro-pathic families. Hysteria in the mother is very frequently followed by hysteria in the daughter. More commonly, however, the transmission is by transference from, or to, other nervous and psychoses. *Artificialism* or *pédisme* in the antecedents of hysterics plays the same part as in other manifestations of degeneracy.

**Inciting Causes.**—*Emotional disturbance* of any sort may initiate hysteria. Fright, grief, worry, elation, and every sort of mental and moral strain and shock are the common starting-points of this multifarious disease. Freud, of Vienna, has laid great, too great, stress upon the sexual factor in hysteria and allied psychoneuroses. Errors, shocks, emotions, excesses, abuses and emotions in the sexual sphere are sufficiently common inciting causes, but by no means the only or even the essential ones. Traumatism furnishes a large quota of hysterics, especially of the male sex, owing to their greater liability to such accidents. In all such circumstances, unless consciousness be abolished instantly without preceding

anxiety or fright, the attending psychic states must be taken into consideration. As a practical fact, the likelihood of hysteria following trauma is in direct proportion to the intensity of the mental shock. The physical injury may be insignificant. Lightning-stroke, surgical wounds, and internal conditions, such as gastric ulcers, nephritic and hepatic colics, may act as causes.

*Intoxication* by lead, mercury, sulphid of carbon, oxid of carbon, tobacco, morphia, cocaine, and chronic alcoholism, or even a single alcoholic debauch, may induce hysteria. In many such cases these intoxications furnish a basis on which hysteria develops by the incidental action of some other provocation. *Infectious diseases*, such as typhoid, diphtheria, influenza, pneumonia, scarlatina, malaria, and syphilis, may provoke hysteria. It may occur in *convalescent states* due to chlorosis, diabetes, phthisis, and cancer. It is found as an *associate* of all organic diseases of the brain and spinal cord, frequently appearing in tabes, syringomyelia, and insular sclerosis. Either mental or physical *overwork* may cause it. Wherever people of suitable age are domiciled together, hysteria may become epidemic through the force of imitation and suggestion arising from an initial case of hysteria or of some physical disease. Schools, prisons, barracks, and large families may thus become affected. Usually in such instances there is great similarity among the cases. In this country, under the prolonged excitement and fervor of protracted religious meetings in rural districts, epidemics of hysterical spasms and even of dancing, in all respects similar to the medieval epidemic dance of St. Vitus, have developed. Hysterical patients in hospitals may closely *imitate* all the symptoms and physical disabilities of other patients with whom they are kept in contact. In other cases they reproduce the manifestations of some disease with which, in their past experience, they have been made familiar.

**Symptoms.**—The innumerable symptoms of hysteria, to follow the plan of the French writers, may be divided into two major groups: those which are essentially persistent,—the *stigmata*; and those which occur incidentally, are intermittent or transitory,—the *accidents* of hysteria. The stigmata are not necessarily present singly or in combination, but, once developed, tend to persist so long as the affection lasts. The accidents present the greatest diversity in different patients, but usually, if they occur repeatedly, tend to uniformity in a given case. Further, some hysterical accidents, as paralysis or contractures, may be of long duration, and, once thoroughly established, have the force of stigmata. Although the cases of major hysteria are comparatively rare it is only through a familiarity with the entire range of the disease, with all the details of the picture, that larvated, partial, and fractional states can be recognized. This is particularly true of the temporary or incidental features of this disease, and especially of the convulsions.



## STIGMATA OF HYSTERIA.

The stigmata of hysteria are sensory, motor, and psychic.

**Sensory Stigmata.**—In hysteria the sensory disorders are (1) of the negative variety,—hypesthesias, anesthetics; and (2) of the positive sort,—hyperesthesias. They are usually both represented in a given case, but the anesthetics are the more important symptomatically. Owing to the heightened suggestibility of hysterics, merely searching for sensory changes may inaugurate or modify them, but even so their presence has its symptomatic value.

**Hysterical anesthesia** may affect sensation in all its modes and senses, including the special senses. The general cutaneous sensibility may be disaggregated so that only certain elements persist and a limited group of stimuli alone serve to arouse the sensorium, as in a thermo-analgesia that parallels the sensory dissociation of syringomyelia. The diminution of sensibility may be partial or complete, and often varies in intensity and area in the same patient within a very short time. Various degrees of anesthesia may likewise be found in different regions, and the anesthetic area represents remarkable variations of extent and distribution in different cases, and also in the same case at different times. Some form and degree of anesthesia is rarely lacking in hysteria that has existed any length of time, and often it is developed very early. It is obligatory to persistently search for it in every instance, but care must be exercised not to induce it by suggestion. As a rule, hysterics are themselves ignorant of their sensory deficiencies. The anesthesia may be (1) superficial, affecting mainly the skin and mucous tissues, or (2) it may involve the deeper structures.

**Cutaneous anesthesia** may be absolute. Pricking, pinching, hot and cold bodies, produce no response. Some patients are merely analgesic, and this is the common defect. Less frequently there may be thermo-anesthesia or thermo-analgesia, and, most rarely of all, tactile sensations alone may be wanting. Very exceptionally the hysterical patient feels the faradic current in the anesthetic area or, more rarely still, presents an area insensitive to this stimulus alone. Complete anesthesia, hypesthesia, and hypalgesia are the commonly encountered forms. The successiveness within the range of examination may show the same anesthetic modifications of sensibility. The buccal, pharyngeal, laryngeal, nasal, conjunctival, anal, urethral, and vaginal surfaces may be entirely insensitive or present dissociation of sensation. In the area of cutaneous and mucous anesthesia there is usually, if not always, a modified vasomotor change, so that pricklings which readily draw blood elsewhere do not bleed.

The deeper parts are frequently anesthetic. Bones, muscles, ligaments, and nerve-trunks may sometimes be pierced, twisted, wrenched, and contused without giving rise to distress or even provoking a localized sensation of any sort. The *susceptor* sense for a limb may be abolished, so that with bandaged eyes the patient has no knowledge of its position, cannot estimate weights, recognize pressure, or feel fatigue.

**The Special Senses.**—Taste and smell may be perverted, diminished, or abolished. Certain solid articles may fail to arouse the sense of taste, while others are still detected. The loss of taste is usually limited to a portion of the tongue and mouth. Hearing is often greatly diminished, but complete hysterical deafness is very uncommon. Rinne's test (see p. 65) shows the disturbance to be central. When the loss of hearing is pronounced, one commonly finds more or less anesthesia in the auditory canal and on the same side of the head and face, but absolute anesthesia of the drumhead is exceptional.<sup>1</sup>

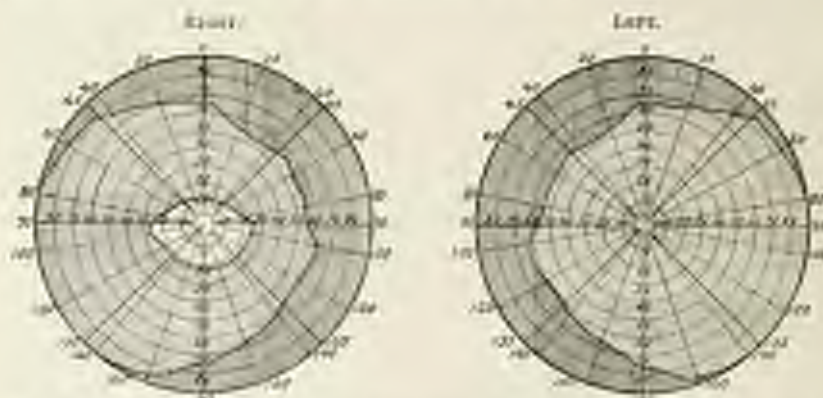


Fig. 246.—Hysterical concentric contraction of visual field of right eye; anastropia of left eye (Tourneux).

In hysteria vision is very frequently modified, and some of the changes in this special sense are of the utmost importance for diagnosis. Complete blindness is very rare, usually of abrupt onset, a few days' duration, and sudden recovery, but vision is often reduced in one eye to counting fingers or less. Of greater frequency and of more importance are the lesser and commonly persistent defects. These consist of: (1) A reduction of the field; (2) troubles of color perception, and (3) errors of accommodation.

**Contraction of the Visual Field.**—In the great majority of hysterics the visual field is concentrically contracted. This is usually found bilaterally, but commonly more on one side than the other, and sometimes only on one side. The defect, as in that of all the special senses, when unilateral or most marked on one side, usually corresponds to a unilateral distribution of disturbed cutaneous sensibility, but the opposite situation may be encountered. In total amblyopia the contraction of the field reduces it to zero. In a given case the retraction of the field is practically permanent, but it may fluctuate greatly. An epileptiform attack, fatigue of the eye, emotions, and variations of attention may modify its limits. Hysterical hemiopia and scotomata are so infrequent that they should always suggest an organic lesion.

The *obscuration* is more characteristic of hysteria than the contracted field, which is also present in neurasthenia. In the normal eye the fields for the various elementary colors are not coextensive (see

<sup>1</sup> Chavanne, Paris, 1880. "Oculis et Hysteria."



p. 64). In their natural order blue has the largest field, followed by yellow, orange, red, green, and violet. In hysteria not only are the fields for form and light perception contracted, but those for colors are also diminished, and to a proportionate degree. The important particularity is that the normal order is generally changed. The *red field often exceeds the blue*. When the color-fields are extinguished, they usually disappear in this order: violet, green, blue, yellow, and, *last of all*, red. Objects are then finally seen as grayish, and we have the condition of achromatopsia.

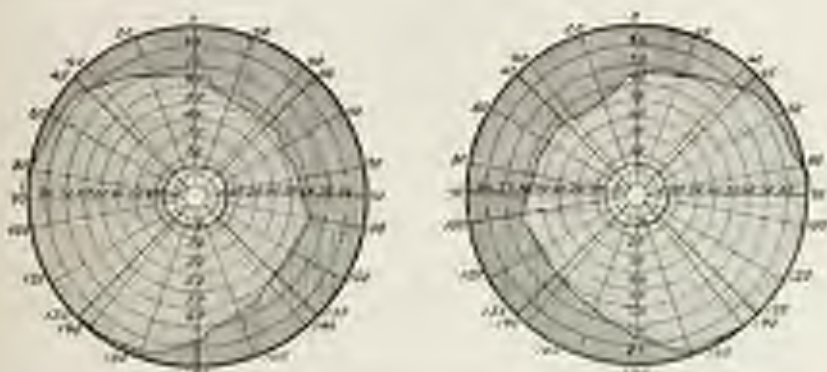


Fig. 20.—Hypothetical bilateral concentric contraction of visual fields (Tausch).

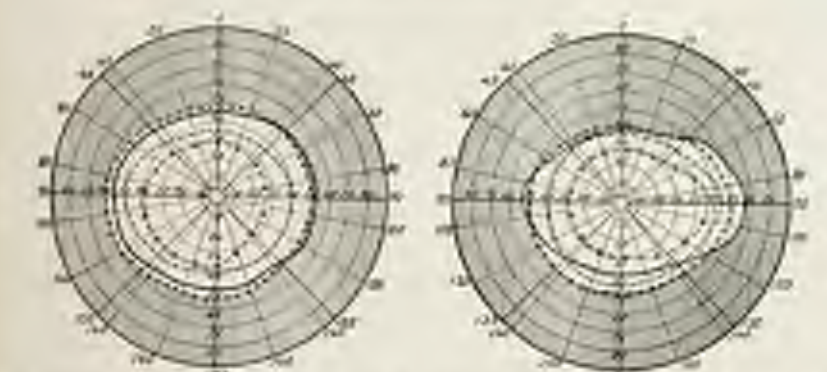


Fig. 30.—Concentric contraction of visual fields for colors usually found in hysteria. Red field (solid line); — — — — blue field; ..... green field; x — x — opaque.

*Accommodative errors* in hysteria are sometimes encountered, by which near vision, particularly, is rendered faulty. To the same source have been attributed the *accommodative diplopia*, *polyopia*, *monoplopia*, and *microplopia* that are occasionally met. In this condition, with the opposite eye covered, the patient fixes the gaze upon a pencil close to the eye. As it is withdrawn, its image doubles, and at a greater distance a third may appear, and they all increase in size, and again diminish as the pencil approaches. Errors of curvature in the cornea must be excluded to render the symptom significant.

It has recently been recognized that, aside from complete bilateral blindness, the various forms of hysterical amblyopia are not present in binocular vision. Even opening the unaffected eye, in the case of partial unilateral amblyopia, at once widens the contracted field (Parinaud). Patients who cannot read with either eye alone may do so readily with both. That the amblyopic eye sees is easily demonstrated. With a red glass over the affected eye a prism over the other doubles the image of a flame, and the red image, corresponding to the anastrotic eye, is distinctly seen by hysterics. Or, in a case of unilateral green-blindness, a prism doubles the image of a green object and both are perceived as green. Again, an eye that fails to detect everything but red, upon looking at a revolving Newton wheel bearing red and green, sees it to be whitish, as does the normal eye, proper cognizance having evidently been taken of the complementary green. It is evident, therefore, that the trouble is not in the eye or in the paths of conduction, but that, under ordinary circumstances, the impression fails to enter the field of personal consciousness. These symptoms are unattended by any change in the retina or media. Spiller<sup>1</sup> insists that the *lacrimal reflex* remains in hysteria even when the eye-lids and the side of the face are anesthetic, while in organic lesions the tears are not similarly secreted upon irritating the conjunctiva, as with a piece of paper placed under the lid.

**Distribution of Hysterical Anesthesia.**—In a very small proportion of cases the anesthesia in hysteria involves the entire cutaneous and mucous extent. Usually it is limited (1) to one-half of the body; (2) to areas of more or less definite geometric outline; and (3) to discrete idets.

*Hysterical hemianesthesia* is a common distribution of sensory deficiency. Ordinarily, it affects the left half of the body, and is sharply limited by the median line. If intense, the approachable mucous surfaces on the same side are commonly also anesthetic. As a rule, the special senses—sight, hearing, smell, and taste—are blunted on the anesthetic side, but in some cases the special senses are affected on the opposite side.

*Anesthesia in geometrical areas* is frequently encountered. An entire extremity or a band about an extremity may alone be anesthetic. The distribution may often be described as a sleeve, glove, sock, stocking, or drawers-leg anesthesia. The outlines have no relation to the anatomical distribution of the nerve-trunks whatever, but are referable to functional groupings. They often have a superimposed relation to functional disturbance, as in cases of paralyzed or contracted limbs, diseased joints, or local injury. In hysterical amblyopia the conjunctiva, lids, and a circular area about the eye are usually anesthetic. In hysterical deafness the auditory canal and eardrum are frequently insensitive. In hysterical aphonia the larynx may be anesthetic.

*Idets of anesthesia* of peculiar and paradoxical outline are sometimes encountered. These may be many or few, and it often requires minute and painstaking search to find them.

<sup>1</sup> "Paia. Med. Jour.," May 17, 1902.



**Peculiarities of Hysterical Anesthesia.**—1. In the first place, the outlines of limited hysterical anesthetics, excepting hemianesthesia, do not conform to the anatomical distribution of cutaneous nerves or to the sensory areas related to the vertebral segments, but rather to the mental association of functions, and probably to their cortical representations.

2. The sygnic and trodden reflexes in hysterical anesthesia are not modified, as in organic lesions marked by insensitiveness. The pupil, as a rule, responds to light and accommodation and to pinching of the neck.



Fig. 252-1. Most common distribution of hysterical anesthesia. (a) and (b) Hemianesthesia (Duché). (c) Hysterical anesthesia of prominent regions (Toussier). (d) Hysterical anesthesia of insensitiveness (Duché). (e) and (f) Complex patterns (Duché).

even when the eye is amblyopic and the skin of the neck is insensitive. The abdominal, cremasteric, knee, and toe reflexes are not abnormal. The cardiac and respiratory reflexes to painful cutaneous stimulation are retained, even when an anesthetic zone is used. Sometimes a spurious ankle-clonus can be obtained. Other reflexes dependent principally upon sensation are abolished by hysterical anesthesia. The pupillary and pharyngeal reflexes disappear if the parts are insensitive. The pharyngeal reflex is lost in 90 per cent. of hysterics. Tickling of the

trunk or the plantar surface does not produce the usual responses. Buzard lays especial emphasis on the loss of the plantar reflex, with retention of the knee-jerk, in hysteria.

In many instances the knee-jerk is relatively increased, and often a slight clonus may be obtained at the ankle. There is also a tendency for a tap on the knee to induce jerking of the trunk or both limbs, in addition to the true knee phenomenon.

3. The anesthetic zones are movable. Though they may persist for months, and even years, they are not absolutely fixed, and often are even capricious. A number of things may serve to promptly or gradually modify them. Hysterical attacks frequently change the distribution of the anesthetic zones, and sometimes are preceded by an increase in their size. During such attacks the anesthesia may completely disappear. Anesthetics or a dose of morphin or alcohol sometimes cause its temporary disappearance. Hypnotic somnambulism and suggestion may displace anesthetic areas, and they may disappear during sleep. If the patient's attention be strongly fixed on an anesthetic area, it may momentarily restore sensation. In the same way, as pointed out by Patrick, the boundaries of the anesthetic area may enlarge during the course of an examination, the attention in this case naturally being directed to the loss of sensation. The action of so-called *orthogenetic agents* is supposed to be largely due to the attention and suggestion they invoke. By the application to the anesthetic areas of a great diversity of objects, such as metal plates, magnets, woods, metals, minerals, and gases, or by the use of electrical currents, especially faradism and static sparks, the anesthesia may be made to shift, sometimes to disappear, and often to *transfer* to the opposite side. Some patients are susceptible to one agent, some to another, apparently in proportion as their attention or fancy is captivated. When the anesthesia is transferred, it tends to return to the side whence it was displaced, but in doing so oscillates from side to side several times before finally locating in its old habitat. A *transformation* of tactile into visual sensation sometimes occurs. Binet first described this curious phenomenon. If letters, figures, or geometrical outlines be traced with the finger-nail or a pencil upon anesthetic skin areas, the patient can see them on a blank wall or screen. Fry<sup>1</sup> also found that if a colored screen were used, the patient described the tracing as surrounded by the color complementary to the one before her; for instance, with a blue screen the figures appeared to be in a red field.

4. It is a striking and paradoxical fact that hysterics are not disturbed by their sensory losses, and are usually entirely ignorant of them until deciphered by a medical examination. For instance, the small and delicate movements of the fingers ordinarily so dependent upon sensation may be perfectly performed by the anesthetic hand of the hysteric under the guidance of the eye. Apparently the motor image is called up and exteriorized through the motor paths.

5. It has been noticed that a hand completely anesthetic to all forms of sensation would yet grasp a pencil or other familiar object, and promptly indicate its use, when the patient's eyes were bandaged and all sensation denied. In the same way an anesthetic leg and foot may

<sup>1</sup> "Jour. Mental and Nervous Dis.," Aug., 1899.



cause no incoordination in walking. These paradoxical features have usually been attributed to deceit and simulation, but are presented by similar cases the world over without possibility of collusion.

6. The sensory disturbance is frequently located by topical or traumatic causes. The frequency of left-sided hemianesthesia is related apparently to the lesser strength of the left side. The superposition of sensory disturbance to inflamed joints or to injuries or to points of focused attention may often be noted.

**Hysterical hyperesthesias** are very common. Neuralgic pains and other disturbance of sensation may occur in hysterics as well as in others, without having any special significance, but often from such a source or from a blow or from the mere fixation of attention, perhaps through misdirected solicitude of the physician, there develops a *peculiar sensitiveness*. This is usually circumscribed. It may involve a joint (Bosley's joints) or an entire limb, but is practically never generalized or even of hemiplegic distribution. It is often confined to narrow, superficial zones or points, as at the vertex, giving rise to the classical *clonus*, about the breasts, along the spine, in the groins, and at the pit of the stomach. The glandular portions of the breasts, testicles, and ovaries may be similarly sensitive. Such sensitive spots are frequently found in the midst of anesthetic areas, and while the surface may be exquisitely sensitive to the touch of a finger, the pricking of a pin may cause no discomfort. It will frequently be found that if the patient's attention is strongly diverted, the area so intensely sensitive may be less sensitive than normal and entirely tolerant of firm and deep pressure. In the same way, by suggestion or the application of various inert substances, the sensitiveness may be, at least momentarily, suppressed. There is always found, upon close investigation of such sensitive areas, paradoxical peculiarities similar to those in the anesthetics, and which serve to proclaim their hysterical nature.

If such a hyperesthetic zone arises from or becomes associated with some mental storm, pressure upon it may serve to revive the memories in question and provoke a hysterical fit. It is then denominated a *psychogenic or hysterogenic point or zone*. The mental character of hysterical hyperesthesias is evident in their genesis and associations. Of a similar nature are the disagreeable or painful sensations provoked in rare cases by certain substances or by heat or cold. Paresthetic tinglings, numbness, etc., are comparatively rare.

**Motor Stigmata.**—The motor stigmata of hysteria must be sharply distinguished from the motor accidents to be considered later. The stigmata are usually unknown to the patient, and must be sought by the physician. Practically, they differ from the accidents only in degree, and show the substratum of more marked errors of motor control, which are likely to develop as accidents at any moment. In many ways they are indicative of the automatism which is one of the most marked features of the psychoneurosis.

1. **Movements in hysteria are retarded.** The reaction time is increased often for the simplest movement, and in proportion to the insensibility of the parts brought into play. The retardation, therefore, is closely allied to the anesthetics. It may be increased by diverting the patient's attention or diminished by concentrating his attention upon

the given act. In automatic movements provoked subconsciously or under hypnosis the reticulation disappears, showing its psychical nature.

2. Movements are maladroitt and incoördinate, unless carefully supervised by the patient; and this, again, is proportionate to the anesthesia and the obliteration of the muscular sense. In automatic and subconscious movements the incoördination disappears. Partial or complete catalepsy and automatism in such limbs may be present if the patient can not see the limb or otherwise gain an idea of its position.

3. Hysterical patients are often incapable of performing several acts simultaneously, as they are unequal to the division of attention thereby necessitated.

4. Voluntary intentional movements are usually weakened. Patients who may in an automatic manner show great strength in the performance of customary and habitual tasks, when asked to make a test effort, as upon the dynamometer, register an insignificant amount. They appear incapable of willing an effort of which they do not possess the deeply graven motor image. The difficulty again is mental—the outcome of their diminished volition and attention. Anesthesia is most marked in the insensitive parts, and foreshadows the paralytic accidents.

5. In many hysterics there is a tendency to rigidities or contractures. It may be demonstrated in weakened or anesthetic limbs, and is often indicated by exalted reflexes. It may be provoked by an elastic bandage, deep massage of the muscles, sharp flexion or extension of joints, fardication, frictions, persuasion, and suggestion, varying in different patients. The contractures thus induced invade both flexors and extensors, and fix the joint in a characteristic attitude, the same as that in the transient and spontaneous hysterical contractures. By similar means it can be caused to subside. Charcot denominated this state the *dialéxis of contractures*.

**Mental Stigmata.**—The mental stigmata of hysteria consist in a belittlement of memory and of will-power. The amnesia is sometimes due to the lack of mental concentration,—itself the outcome of the enfeeblement of volition,—and the loss of will-power is also frequently manifested in the impulsive acts and general want of self-control.

1. *Amnesia.*—The forgetfulness of hysterics accounts frequently for their uncertain and contradictory statements, and has often unjustly laid them open to charges of deceit. In some instances the memory loss is *episodic*,—that is, it embraces a certain group of related facts pertaining to some person or event, while other contemporaneous incidents are recalled. In the same way a group of motor images may disappear,—such, for instance, as those for walking or writing or for articulate speech,—and *ataxia*, *agraphia*, and *motor aphasia* result. In this way the recollection of a certain person may completely drop out of their minds, or they may lose all the words of an acquired language. In other cases the memory loss may be *focalized*; that is to say, it embraces a given period of time. Frequently after a convulsive attack, sometimes in traumatic cases after the initial accident, there is a loss of memory for a variable period of time antecedent to the incident in question, or for a period both before and after the mental disturbance. Rare instances are recorded in which the amnesia has been *total* and complete for all acqui-



sitions up to or during a certain period of life. Such patients begin again to speak and learn as children. In some cases acts and impressions are forgotten immediately. A book or a short story may be read repeatedly with full interest and appreciation, but is at once forgotten. In all marked cases of hysteria there is some blurring of recent memories.

The amnesias of hysteria are analogous to the anesthetics, and, like them, under the action of hypnosis or in subconscious states, may be demonstrated to be purely functional. The memory impressions do exist, and they can be revived. Recovered cases similarly regain the faculty temporarily lost. Out of localized amnesias a *double personality* may arise, as under similar circumstances the hysteric loses one group of memories and regains the other, alternating between the two.

*Morbid* implies absence of will-power. It expresses the impaired volition of hysterics, which reduces their powers of mental concentration and attention, and renders them vacillating, impulsive, and lacking in determination. In some cases it attains such prominence that they can not bring themselves to undertake the simplest task, such as dressing or undressing, and hesitate at the slightest obstacle.

*Impressionability* or *suggestibility* is often extremely developed in hysterics, and practically constitutes a mental stigma. They are emotional and easily moved, subject to the slightest influence and sensitive to insignificant impressions. The lack of will-power renders them of infirm and vacillating judgment, so that they often become dependent upon others to decide their slightest actions. The insistent, underlying, fixed idea thus controls them the more thoroughly.

*Simulation*.—From inadequate knowledge of the disease, hysterics have been supposed to simulate for the pleasure of it, and to deceive for the satisfaction derived from notoriety. To a very slight degree this may be true, but the simulation and the deceit have their origin in misconceptions, in misunderstood impression, or arise from the failure of impressions to reach the field of consciousness. Hysterical young women are too commonly supposed to be craft. This is sure to be the case if in their attacks they show emotional attitudes or actions that may be so interpreted. As a matter of fact, they are usually timid. Introspection and self-concentration are fatal to the grand passion. Local anesthetics or hyperæsthetics may completely destroy the generic sense. It remains to be said that some hysterics do simulate purposely, and even cause wide-spread and dangerous lesions to maintain interest and sympathy. No fluctuation of temperature, skin lesion, or ulcerative process should be considered hysterical unless its fictitious production can be absolutely denied.

#### ACCIDENTS OF HYSTERIA.

We come now to the more or less transitory features of hysteria, the accidents, chief among which are the hysterical attacks. These often take the form of severe, intense, and prolonged convulsions. While uncommon in this country, they are occasionally observed in the most typical form. Ordinarily, however, the attack is partial or hazy. Only by an understanding of the extreme variety will we be able to estimate the simpler forms, which usually consist of isolated elements of the typical *grande attaque* of Charcot.

**Hysterical Convulsive Attacks.**—The complete grand attacks, as studied and described by Charcot and figured by Richer, are infrequent, but in some irregular or fractional form they occur quite commonly. Patients possessing spasmogenic zones may usually be thrown into a convulsion by firm pressure on these points, and during the seizure similar pressure again commonly causes it to subside. Emotional disturbances may cause the attacks or they may apparently come on spontaneously. The descriptions of the Charcot school and the delineations of Richer were based on a definite group of cases which were in a sense cultivated and fostered by association, interested study, local and medical suggestion, and some preconceptions. Such cases are no longer seen at the Salpêtrière in a typical form, but they serve to show the extreme to which the condition may go and embrace all the elements of more usual and ordinary cases. It has been considered wise to retain their general outlines.

The grand attack consists of a premonitory stage, followed by four periods: (1) The premonitory stage varies in different patients, but is uniform for the given case. Some patients are depressed, taciturn, and moody; others exuberant, restless, quarrelsome, and talkative. Many have hallucinations of sight or of hearing, referred in the direction of the anesthetic side, and the insensitive areas may be increased. Usually, palpitations and vasomotor storms are observed. There may be nausea, hiccup, trembling, and the discharge of a large quantity of urine. The scene follows. This, ordinarily, consists of a painful feeling arising in the lower part of the abdomen, and develops into the sensation of a rounded body, which mounts upward, and, as it reaches the neck, gives rise to feelings of strangulation or suffocation—the *globus hystericus*. The face may flush, hissing is heard in the ears, throbbing is felt in the temples, objects turn dark before the eyes, vertigo occurs, and the patient sinks down, or even falls suddenly, unconscious, and the fit begins. In many instances the fit does not develop, and in a moment the patient is relieved, or the globus may last for hours without eventuating in a fit. Sometimes this train of symptoms is incited by emotional disturbances arising from insignificant irritation, or any strong mental impression may induce it.

2. The *qualeptoid period* closely mimics the features of an epileptic attack. There may even be an initial harsh noise or slight cry. The patient is rigid; the face is pale, but promptly becomes red, and the neck is congested and swollen. Frequently the tongue is protruded at the teeth may be ground together. Biting the tongue and involuntary urination are uncommon, but do occur. Usually the convulsion is most marked on the anesthetic side to which the face is turned. The *tonic phase* lasts two minutes or less, and is often attended by slow, rigid movements of wide range, with notable extension of the feet and supination of the hands or movements of circumduction, unlike anything seen in epilepsy. The fingers are usually clenched over the thumb, which may protrude between them; the chest and abdomen are fixed and the body is rigid. This tonic phase is followed by a *clonic phase*, in which rapid, small oscillations begin in the rigid members and in the face. The suspended respiration reappears in broken, arrhythmical gasps and sobs, the chest and abdomen acting independently. Noisy movements of swallow-





Fig. 252.—Tonic phase, the tongue rolling from one angle of the mouth to the other (Eckart).



Fig. 253.—Tonic representation of the whole tonic movement (Eckart).



Fig. 254.—Tonic phase, circumduction movements of upper members (Eckart).



Fig. 255.—Clonic phase, tonic representation of clonic movements (Eckart).



Fig. 256.—Phase of convulsion (Eckart).

ing and sensorius lortorygni are frequently produced. All the clonic movements are independent and illogical. This phase may last several minutes and gradually subside, the patient falling into a phase of *remora-*



Fig. 25.—Phase of tonic contraction, retaining partial contractures (Hickey).



Fig. 26.—Piked position tonic (Hickey).



Fig. 27.—Tonic posture tonic (Kendrick).



Fig. 28.—Tonic posture tonic (Hickey).

tion, in which some rigidity may, however, persist. Stertor and frothing saliva may appear, and sometimes general twitchings or sharp spasms



may still agitate the patient at intervals. The pupil may not respond to light.

3. *The Period of Clonus.*—After the phase of resolution of the epileptoid period has lasted a short time, the second period develops. It is made up of two phases,—(a) a phase of *convulsions*, or illogical attitudes, and (b) a phase of *wild-raged*, or *grand, convulsions*. The convulsions of the first phase are usually steadily maintained for several minutes, thereby varying essentially from the grand movements of the



Fig. 361.—Postural attitude of struggling with an assistant (Michez).



Fig. 362.—Postural attitude of withdrawal (Michez).



Fig. 363.—Zappa (Kocher).



Fig. 364.—Decline of the fourth period (Kocher).

second phase, which are repeated with more or less rapidity. One of the most common and characteristic convulsions is an exaggerated *opisthotonus*, or *arc de cercle*. This may also be executed forward or laterally. To these convulsions succeed alternate flexions and extensions of the trunk or of the limbs, or rotations of the head. This phase is attended usually by violent catarrhes, and, in evident fear or rage, the patients tear their garments, groan in a horrible manner, and put forth an astounding amount of strength against those trying to control

them. In this phase they often bite, scratch, and strike at their attendants, apparently under the domination of the hallucinations of a fixed dream or delirious storm.

4. *Period of Passional Attitudes.*—The third period is the gradual outgrowth and logical continuation of the second. The patient dramatizes in pantomime the acts of the dream that embraces circumstances of the past life, or perhaps the incidents connected with the origin of the hysterical condition. Terror, love, gaiety, rage, eroticism, singly or by turn, animate the features and control the attending attitudes and gestures. The acts, emotions, and attitudes of one attack are usually repeated with fidelity at each succeeding crisis by a given case. If one knows the nature of the dream, it is easy to anticipate its manifestation.

5. *The period of delirium* is a prolongation of the dream state of the third period. It still pursues and dominates the patient, who now talks in the delirium and verbally expresses his hallucinations, which usually have to do with disagreeable sights, animals, and acts. The punctum, in which red often predominates, unrolls from the anesthetic side. The passionai attitudes may be occasionally repeated. The delirium may be gay, furious, sad, or obscene, and the language and actions correspond. After a varying time the hallucinations fade, and memories may recur, with sobs and tears, and suddenly, or after a few moments' silence, the patient awakes, a little fatigued, and at once fully recovers his former conscious state.

The duration of a grand attack is variable, but on an average requires from fifteen minutes to half an hour. The prodromal stage and the fourth period may be very long. The epileptoid period rarely exceeds three minutes; the second period ordinarily lasts five to ten minutes; the third is still longer. In some instances one attack follows another without appreciable interval, or some feature of the attack, as the stupor of the first period or the delirium of the fourth, may be prolonged for hours and days, constituting a *status hystericus*. The following table may serve to systematize the features of the grand attacks:

Premonitory stage:	Profuse.	Mental disorders and hallucinations. Functional organic disorders. Motor and sensory disorders. Aura, globes.
Epileptoid period.	Onset. Tonic phase. Slow movements and tetanic rigidity. Clonic phase. Small, quick movements. Phase of resolution. Sopor, stupor.	
Period of clonism.	Phase of convulsions, arm de circle. Phase of large movements, sublim.	
Passional period.	Passional attitudes and actions.	
Period of delirium.	Delirium, hallucinations, somnia.	

**Modified or Partial Attacks.**—The typical grand attack may be infinitely modified. It may be intensified or reduced in severity, but, what is of more importance, it may be disaggregated, and almost any feature of it may occur alone as a partial attack. These partial attacks



may also be indefinitely prolonged in a condition of status. Some of the most commonly encountered ones follow:

*Vertiginous Attacks.*—The premonitory vertigo, obscuration of vision, ringing in the ears, and sinking feeling may recur repeatedly, without other features of the attack.

*Globose attacks* are among the most common of the larvated seizures. The patient suddenly feels the discomfort at the throat, or it may slowly appear, and only disappear after a considerable period.

*Epileptoid Attacks.*—The attack may cease with the phase of resolution belonging to the first period, or, recurring at this point, strongly simulate the epileptic status. The differential diagnosis is often extremely difficult, the more so as epilepsy may affect a hysterical subject, and convulsive manifestations of both neuroses may alternate.

*Idiotic Attacks.*—The seizure may be limited to the first phase of the second period. The *arc de cercle* suddenly appears, and may persistently endure, or to it may be added the clonism, with wide movements, giving, for instance, salami attacks. Rage, joy, and other emotions may then modify the movements and the attitudes, and demoniacal or maniacal attacks are presented, in which the patient may be extremely wild and unmanageable.

*Attacks of ecstasy* are manifestations of the passionai attributes of the third period. With or without premonition or aura, the patient suddenly becomes motionless, and the face expresses astonishment, admiration, adoration, or stupor. Cataleptic features are often added.

*Syncopeal Attacks.*—After an aura or some feeling of discomfort the patient relaxes and becomes inert and apparently unconscious. The hands may be clenched or a tremor of the eyelids may indicate the spasmodic state. Respiration slows, the face may be pale or slightly flushed, but the pulse is unaffected. This attack may be prolonged into the sleep attack.

*Attacks of Sleep.*—This lethargic or trance-like condition may follow several ordinary attacks, or some emotional storm may induce it. Its onset may be gradual, or sudden and apoplectic. The appearance is one of natural sleep, but careful search will often detect slight contractions, as in the eyelids, which do not easily yield to the finger, in the ocular squint, or the position of the hands and feet. Frequently there is a marked tendency to catalepsy, and the limbs retain any position passively given them. The pupils are sensitive to light, and, though general sensation seems abolished, it is only masked, as such patients frequently report all that has occurred during the sleep. The pulse-rate, ordinarily normal, may be diminished or increased. The respiration is usually shallow and reduced in frequency. The temperature may be normal, subnormal, or slightly elevated. Such attacks may last minutes, hours, or months, and terminate commonly by a convulsive attack, by laughter or tears, or a few delirious words.

*Somnambulic Attacks.*—Some patients, under a sudden impulse, start on long flights or make considerable journeys, in which they may travel great distances and consume many days. Finally, they come to themselves in great surprise at being from home, and may have no rec-

ollection of the intervening events. In a succeeding attack they may recall everything that transpired in the first, and, by a repetition of such protracted attacks, build up a double existence or personality, or the morbid state may eventually completely displace the normal one. In lessened degree automatism and somnambulic attacks of short duration are not uncommon in hysteria.

**Motor Accidents.**—Paralyses and Contractures.—Both the paralyses and contractures of hysteria are foreshadowed by the stigmas of anæsthesia and the diathesis of contracture, of which they may be considered temporary exacerbations. They are frequently combined in a given patient, and even in a given limb. They may follow: (1) Convulsive attacks; (2) mental impressions or shocks; (3) trauma; and (4) various morbid states. It happens that after a convulsive attack a hemiplegic, paraplegic, or monoplegic palsy supervenes and tends to persist for a considerable time. The other exciting causes mentioned act with less rapidity, and there is usually, even in traumatic cases, an intervening period of freedom from motor difficulties,—a period of modulation, as Charcot designated it, or of autosuggestion,—lasting a few hours or several days. In addition, it is to be noted that there is no necessary relation between the severity of the physical exciting cause and the extent or completeness of the paralysis and contractures. They are relative, however, to the mental shock.

*Paralyses of hysteria* are commonly marked by a sudden emotional onset, or they may gradually develop after some such moral storm. Rarely do they abolish every movement in a limb, and close observation will usually detect slight evidences of voluntary or automatic motility. The antagonistic muscle-groups are equally affected and the limb is quite limp. There are vasomotor and trophic disorders in very exceptional instances only. The local temperature may be slightly reduced, and edema be encountered. Electrical and tendon responses are normal. There may be superficial or deep anæsthesia or hyperæsthesia. Variations of the extent and intensity of the paralysis follow various influences or occur spontaneously. Ordinarily, hysterical palsies terminate in complete recovery, but may last days or years. In some instances they are succeeded by contractures, or palsy and contractures alternate. In rare instances the paralysis is clearly identical, in that it occurs only for certain groups of voluntary movements, such as those of writing or walking.

*Hysterical contractures* present loss of power, with persistent involuntary rigidity, without modification of the electrical or tendon responses. It is a rigid palsy. The affected limb is more or less rigid, the muscles are tense and firm, and the contractures persist during sleep, but yield completely during general anæsthesia. Trophic disorders are rare, except in cases of several years' standing, when the muscles may be changed by actual fibrosis and permanent surgical deformities result. Ordinarily, there is superficial or deep anæsthesia, or superficial sensitiveness. In slight cases the contractures may subside during sleep. Rare cases present exquisite sensitiveness and severe paroxysms of pain. Commonly the onset and termination are abrupt, but may be gradual.



Contractures, once developed, show a tendency to protracted duration. They impress characteristic attitudes upon the limbs. The arm is usually abducted; the elbow, wrist, and fingers forcibly flexed. The hand, when alone affected, may be flexed into a fist, with thumb under or between the fingers, or the hand may be extended or held in the writing position. The lower extremity is commonly extended in all its joints. Rarely, the foot is dorsally flexed. The muscles of the trunk are not infrequently contracted, causing deviations of the spinal column mimicking scoliosis and anterior and posterior curvatures. The toes may be extended or flexed. Many of these attitudes are transiently represented in the hysterical attacks, as shown in the figures on pages 631 and 632, and in some sense hysterical contractures or palsies may be considered prolonged localized attacks, or as a hysterical status of fractional extent.

Some of the more common varieties of hysterical motor accidents follow.

*Hysterical hemiplegia* affects either side with about equal frequency;



Fig. 267.—Hysterical contracture of foot and leg.

Ernest Jones<sup>1</sup> in a tabulation of 277 cases found it approximately 55 times on the right to 45 times on the left side. The distal portions of the limbs are most affected. Instead of walking with the rigidity of organic hemiplegia, the foot and leg are simply dragged along the ground, the advance being made by the sound side. Ordinarily, there is anesthesia of similar hemilateral extent or other sensory stigmata on the same or opposite side. Commonly the face escapes, and, when affected, the lower half is most implicated. Almost invariably limited contractures will at the same time be found, or they may predominate over the flaccidity or alternate with it.

*Hysterical monoplegia* may be single or multiple. Their distribution may be unilateral or crossed. The face and limb on the same or opposite sides, or both lower limbs, or all four limbs may be affected. Usually the paralytic member is not affected in its totality, and presents an anesthesia of greater extent than the paralysis and of characteristic geometric outline. The paretic variety may disturb the urinary control, mainly through the anesthesia of the cutaneous surfaces.

The face may be affected either by paralysis or contracture. In the paralytic variety the upper portion of the face usually escapes, as in brain-lesions. In the spasmodic variety the lips and tongue are most

<sup>1</sup> *Rev. Neurolog.*, 11 March 15, 1903.

affected, and an appearance of palsy on the opposite side is induced as the contracture drags the mouth to its own side. The spasm in the face and tongue is exaggerated by having the mouth opened and the tongue protruded.

*Torticollis* may be due to contraction or to paralysis, and the various position of the head corresponds. Contractures may be confined to the eyelids, giving a false appearance of unilateral or bilateral ptosis. The rigidity and resistance of the eyelids and the depressed eyeballs distinguish it from paralysis of the levator, in which the eyelids are relaxed and the eyeballs elevated.

Contractures and paralyses of the ocular muscles are seldom seen, but do occur. Convergent strabismus may appear. Conjoined movements may be impossible upon voluntary effort, but take place inadvertently. Divergent squint, palsy, or contracture of a single rectus or oblique are not seen in hysteria. Papillary stasis is not an extreme rarity. Karpis<sup>1</sup> contends that in the majority of cases during hysterical attacks the pupil does not respond to light, and that this may also be the case in similar attacks.

*Hysterical contracture* may present all the attitudes of organic hip-disease, though abduction, outward rotation, and apparent lengthening are rare. The apparent deformity subsides under anesthesia, and the joint is found free and smooth. The joint also is not sensitive to percussion over the trochanter, or from the knee. Commonly there is an area of cutaneous sensitiveness, which is bounded by the iliac crest and Poupart's ligament above and a horizontal line below the trochanter. This may also be hysterogenic. Hysterical edema may mimic the local swelling and redness of actual disease, and, indeed, hysterical signs may be added to the true malady.

*Ataxia cerebri* is a hysterical condition characterized by abolition or disturbance of the coördinate movements for walking and standing. In bed or on a chair the patient may show full coördinate control of the legs, but may be unable to stand unsupported, or, if able to stand, is unable to walk. Some such cases, while unable to walk, can run, leap, hop, or climb with ease. The difficulty is due, as already indicated, to a systematized amnesia.

Hysterical rhythmical spasms affect the limbs, face, or neck, causing movements, the same as those purposely executed in health, but steadily repeated with considerable force. They occur in attacks lasting from a few minutes or a few hours to several days. They cease during sleep, and apparently consist of a fractional part of a major attack. Among such rhythmical attacks may be named the *wobbling gait*, in which the head is nodded or shaken or rotated involuntarily, and so forcibly that it is impossible to check it by manual strength. The *choric dance* or *salutary dance*, which played so important a part in medieval epidemics, is of this nature. Some patients move the arms as if using a hammer or other implement, and in general the rhythmical spasms show the dominance of a fixed idea. In a similar way the diaphragm may be affected, causing a peculiar, hoarse, *hacking cough*.

<sup>1</sup> "Jakob. J. Papez," 1888.



*Seizing or grasping* may be repeated in more or less prolonged attacks. In very rare instances the movements may closely simulate the incoördinate, involuntary, arrhythmical action of Sydenham's chorea.

**Hysterical tremors** are of great interest, and often present very difficult diagnostic problems. In hysteria, and as an accident of hysteria, we may encounter all varieties of tremor, not excepting the intention tremor that is so striking a feature of insular sclerosis. Hysterical tremors may be localized or generalized, fine or coarse, rapid or slow, intermittent or persistent, and may last for months and years. The tremor of Graves' disease, of paralysis agitans, of senility, and of metallic poisonings may be exactly counterfeited by hysteria. In some cases the tremor comes on in spells or attacks, in some instances it is limited to the anæsthetic side or to an insensitive member, and in general its exact relation depends upon the sort of hysterical stigmata. The combination of hysteria with the organic diseases which are marked by tremor is very common. This is especially true of multiple sclerosis and the metallic poisonings. Charcot gave the following classification:

#### HYSTERICAL TREMORS.

A.	Tremors, not increased by voluntary movements.	1. Oscillating tremors, three to six per second.	Imitate paralysis agitans or senile tremor.
		2. Vibrating tremors, eight to nine or more per second.	Imitate Graves' disease.
B.	Tremors, occurring at rest or not, provoked or exaggerated by voluntary movements which do not accelerate their rapidly, but augment their amplitude.	1. Intention tremors, five to seven per second.	Imitate insular sclerosis or cerebellar tremor.

Hysterical *tics* may be considered as fractional rhythmic spasms, occurring with more or less irregularity. Like tics in general, they have a purposive character, and are the expression of a fixed though usually subconscious idea. Winking, grimacing, shrugging, sniffing, coughing, movements of the hands, jerking of the legs, which may cause jumping, etc., are encountered.

**Sensory Accidents.**—**Painful Accidents.**—Owing to their more insistent nature the sensory accidents of hysteria are nearly all of a painful character. Sudden amblyopia and deafness have already been mentioned among the stigmata, but may appear transiently as accidents.

*Hysterical cephalalgia* may be deep-seated or superficial, in which case it is marked by a hyperæsthetic zone. In rare instances it is most pronounced in the eyeball. It is likely to occur in periodic attacks, especially toward night, may be attended by vomiting and prostration, and is sometimes intense. It may be confounded with luetic headache, migraine, the pain of cerebral tumor, even with that of meningitis.

*Hysterical pseudowarbling* is occasionally encountered and may

derives the elect. It may present malaise, loss of appetite, pains in the head, increasing to an insupportable intensity and leading to delirium. Vomiting, rigidity of the neck and extremities, fever, vasomotor streaks, and convergent squint may be added. Only a history of antecedent hysteria, an active pupil, a regular pulse, an absence of the dissociation between temperature, pulse, and respiration, and the presence of hysterical stigmata will enable a diagnosis if spinal puncture gives negative results.

*Spinal irritability and tenderness* are frequent, and sometimes constitute the dominant symptoms. The sensitiveness is exquisite, and may be localized over a few vertebrae, simulating Pott's disease, or extend the length of the spine. In many instances it is especially severe over the coccyx. The lightest touch, even the contact of the clothing, may be painful, and the patient can neither lean back in chairs nor lie on the back. Usually, if the patient's attention can be completely distracted, the sensitive zone is found tolerant even of vigorous, deep pressure. It is analogous to the hysterical hyperæsthetic joints and localized hyperæsthesias.

*Visceral neurosias* are often marked by superimposed hyperæsthetic stigmata, and may give rise to the suspicion of appendicitis, gastric or intestinal ulcer or malignant disease, or age the crises of tabes.

*Hysterical organic psychosis* may exactly trace the features of stenocardiac attacks. In some cases it may attend an actual organic lesion, but usually there is no evidence of cardiac disease, and hysterical hyperæsthesia in the precordial region is present. Such attacks are likely to occur in the night, and may have their starting-point in a dream. Often they are preceded or followed by emotional disturbance of a hysterical sort. Generally, the attacks are of short duration, but may persist for several days in a sort of status. The prognosis is not entirely favorable, in view of the fact that it is impossible to absolutely exclude an organic condition during life.

**Visceral Accidents.**—**Respiratory Apparatus**.—Associated with laryngeal paralysis and anesthesia, aphonia is encountered. It usually appears suddenly. The patient may whisper or execute low tones. Sonorous cough generally, and sometimes singing, may remain unimpaired, showing the identical disturbance of vocalization and the systematized defect. Such patients may talk in their sleep with a voice of normal qualities. The laryngoscope may show the vocal bands widely separated or approximated. *Mutism* is more clearly a cerebral defect, a systematized motor-speech amnesia. It may or may not be attended by aphonia. In rare instances *apnoëtic* has been present, but ordinarily the patient writes, gestures, and otherwise readily expresses ideas. The mouth parts usually are entirely mobile and free, but sometimes the tongue is contracted and can not be protruded. Hysterical stammering has also been recorded. Mirror speech is sometimes hysterical. The patient enunciates words backward. Repeated more or less rhythmic cries, *who, bark, hurray, wowing, laughter, grunts*, and various noises are met with, of which the type is the  *hysterical cough*. They are often attended by sensations of a foreign body in the nasal chambers or pharynx, and appear usually in attacks



occurring at various intervals, and presenting a variable duration. They cease during sleep. The cough is very frequent in hysterics of pubescent age, and may appear as the sole manifestation of the disease. A mental, moral, or physical shock may induce it, or abnormal conditions in the nasopharynx may be the starting-point. Acute inflammation or adenoids should be suspected.

*Hysterical dyspnoea* may be (1) of sudden onset, due to laryngeal spasm; (2) it may result from contracture or paralysis of the diaphragm, and (3) it may consist of an extremely rapid breathing, reaching 80 or 100 respirations a minute. The laryngeal spasm is a rare accident, but is very alarming when it does occur, and has resulted fatally. Ordinarily, relaxation occurs when a certain degree of asphyx has been reached. In the form marked by rapid respiration there may be no discomfort or effort, and the pulse is usually normal in rate and character.

*Pulmonary emphysema* and *hemoptysis* are not very rare. Usually there is dulness at the apex, which may strongly suggest phthisis, but lacks the cretation and temperature, and the sputum is free from bacilli.

**Digestive Apparatus.**—Hysterical anorexia sometimes reduces the patient to the lowest level of inanition. Patients reach such a degree of emaciation that they are practically living skeletons, and life is all but extinct. A fatal termination is quite possible. The origin of this state is in some fixed idea of suicide or of expiation. It may even arise from some trivial fear, as of growing too fat. In some instances an esophageal spasm causing *dysphagia* leads to the refusal of food, or it may follow vomiting. Hysterical vomiting occurs in attacks which may last days, weeks, and months. Food is simply regurgitated or violently expelled after a short stay in the stomach. Even fecal vomiting has been observed.<sup>1</sup> Sometimes there is *anuria*, and the gastric ejecta will be found to contain a corresponding amount of uric acid. The amount of vomiting and urinary excretion often alternate in close relation. In another form the esophageal spasm is temporary or more or less prolonged in the form of *globus*. *Typhoides* may appear in association with contracture of the abdominal muscles, and cause a *phantom tumor*, or, if attended by hyperæsthesia, the appearances suggest *peritonitis*. Intestinal spasms are of frequent occurrence in hysteria, and may even produce prolonged obstipation by an *oesophageal contracture*, which is often attended by severe pain and great sensitiveness. It may be mistaken for rectal stricture.

**Urinary Apparatus.**—In addition to the anuria and partial suppression of the urine attending or alternating with hysterical vomiting, and the discharge of large quantities of limpid urine of low specific gravity in the prodromal period of the attacks, or after any of the accidental crises of hysteria, the *masses* and *relative proportion* of some of the urinary constituents may be greatly modified. Tossyette and Cathelinan found that the urine of hysterics in the interparoxysmic periods of their disease was practically normal. Hysterical storms or temporary accidental crises, such as convulsive attacks and the various forms of hysterical status, like rhythmical spasms, delirium, or trance, presented a marked change in the urinary excretion. There is (1) a notable

<sup>1</sup> Bugeau, "Soc. Méd. Centr.," Oct., 1891.

diminution in the fixed residue; (2) the urea is reduced about one-third; (3) the phosphates are decreased about one-half of the normal output for twenty-four hours. Upon examination they found (4) that the normal proportion of the alkaline to the earthy phosphates was altered. Ordinarily, it is in the ratio of alkaline 3 to earthy 1, but immediately after or during such attacks they become about equal. It is necessary to secure a twenty-four-hour collection on which to base such estimates, and it should embrace the period of the hysterical storm. Further, (5) in status lasting several days they observed that the diminution of urea lessened toward the end of the attack, and as it reached the normal amount the status yielded. Although any of the points may be presented in other than hysterical conditions and even in epilepsy, the combination of the first five may be taken as very characteristic. Many hysterics are troubled with frequent urination, which is



Fig. 100.—Dermatographia in a child hysteric.

especially aggravated if the mucous surfaces of the genitals or of the bladder are hyperæsthetic. Mathieu<sup>1</sup> and Balinski assert that polyuria is generally, if not always, hysterical. In short, that diabetes insipidus is a hysterical manifestation, but this opinion cannot be maintained.

*Hysterical fever* may be continued, remittent, or intermittent, and usually, if not always, is found in female cases. After an interval of a few days, or perhaps only after many months, it may disappear suddenly. The temperature may attain a high range—103° or 106° are not infrequent, and 110° and higher have been recorded. Usually the physical state shows no corresponding febrile disturbances, but in some instances the tongue is heavily coated, there is headache, depression, sweats, and general phenomena of fever. Emaciation is very unusual. Commonly hysterical fever is unattended by other hysterical accidents, but it may be associated with pulmonary, meningal, and peritoneal symptoms, and lead to mistakes. The intercurrent form may suggest malaria. In the diagnosis of hysterical fever all sources of infection and hidden suppuration must be carefully investigated and simulation rigidly excluded. Balinski denies the possibility of hysterical pyrexia.

**Trophic and Vasomotor Accidents.**—The trophic accidents of

<sup>1</sup> "Prog. Méd.," Feb. 18, 1899.



hysteria are of recent recognition. They are few in number, and are but rarely encountered in severe degree. Here also simulation must be excluded. Babinski denies all these accidents to hysteria because he cannot produce them by hypnotism. On the skin, erythematous or vesicular eruptions are the most common manifestation, and the vesicular form may go on to ulceration and produce persistent scars. Even cutaneous gangrene has been recorded. Cutaneous hemangiomas in the form of *bloody sweats* have followed severe emotional disturbances. In some historical cases they have given rise to the stigma of the crucifixion or been attributed to the finger-prints of the devil. They are usually preceded by great pain in the parts. *Bloody tears* and bloody discharges from the mammae or from the lungs are of a similar nature. Some hysterics show transitory periods of *dermographism*. *Necrotic ulcers*, which may be red, blue, or white, and *loaf eruptions* similar in appearance to those of Raynaud's disease, may persist for many days, and usually appear in parts otherwise hysterically affected.

The *hysterical breast* is of rare occurrence, but furnishes a source of much apprehension and misapprehension. Suddenly, or within a few hours, one or both breasts enlarge, and are sensitive, painful, and hot. The skin may even be reddened. The nipple is turgid and sometimes erect. The glands are firm to the touch, but not clumous, and the hypersensitiveness is usually extreme. The enlargement may last several days or several months, and, if one-sided, may lead to suspicion of abscess or new growth. When bilateral, the condition has suggested pregnancy, especially as a milky fluid may ooze from the nipples.

*Myotendinous contractures* in muscles the seat of persistent hysterical contractions have already been noted. *Muscular atrophy* has been observed by a number of reliable observers. It has its seat in the muscles of a paralytic or contracted member, and rapidly develops. Within a few days the muscular masses may lose a third or a half of their volume. The condition then remains stationary for a long time, and finally recovers. There is a quantitative loss in electrical excitability proportionate to the muscular shrinkage, but the reaction of degeneration does not occur.

**Course.**—The course of hysteria is essentially chronic. Sensory stigmata have been known to persist for a lifetime, although accidents had long ceased to occur. It should be looked upon as a mental state which is likely to persist when once established, and as constituting a real disability, which may be partial or total. The various accidents may occur transiently, repeatedly, or persist for months and years, and must be individually considered in connection with each case. Under proper management hysteria is usually a manageable disease. Many cases get instant relief under certain mental and moral influences.



Fig. 100. Hysterical contracture and spasm of the hand, lasting several months. Intermittent and relieved. Given used to prevent more serious injury.

**Prognosis** in hysteria is clouded by the probability of recurrence under the action of exciting causes that otherwise might be trivial incidents. Many patients make substantial gain and consider themselves well, when an exhaustive examination discovers numerous persistent stigmata. These may be considered subjective recoveries. It is exceptional for well-developed cases of hysteria to regain absolute health. Children and youths make better recoveries than older subjects. Major hysteria after the age of forty presents very poor prospects of complete recession. Many of the accidents of hysteria and some of the stigmata are capable of instant disappearance, many of the disabling features are easily controlled and dispensed, but the fact remains that the curability of hysteria has been greatly overstated.

**Diagnosis.**—The diagnosis of hysteria poses many difficulties if it is clearly distinguished from neurasthenia, emotional disturbances, and wilful deceit with which it has been commonly confounded. They have nothing to do with hysteria properly considered, but may complicate it. While hysteria is polymorphous, and may mimic all other maladies, in that very fact lies its detection. There is in it always an excessive or paradoxical element. No disease, when well developed, is so distinctly marked and stigmatized. The most reliable and consistent features in hysteria are the mental characteristics and the psychæ stigmata. Next in frequency and importance are the sensory stigmata, among which the disturbance of the color-fields by contraction and inversion is common. Anæsthesia in islets or geometrical outlines are practically demonstrative of hysteria. The mobility or motility of anæsthetic areas under various influences is found in hysteria alone. Among the motor stigmata the contracture diathesis and the ideational loss of power for systematized movements, as shown in astasia or agraphia, while strength is normal in other respects, declare the hysterical state.

Some hysterical accidents are diagnostic. The typical hysterical seizure should be mistaken for nothing else. In the partial and much more commonly encountered seizures, attacks of ecstasy, of sleep, and of somnambulism are very distinctive. A careful examination of the palsies and contractures, taken with the ordinarily associated and usually superimposed sensory stigmata, should disclose their hysterical nature. The rhythmical spasms are the property of hysteria alone. The tremors must be disengaged from the context of hysterical manifestations and confirmed by the exclusion of organic processes. The same is true of the intestinal accidents. In the condition of the urine during and after paroxysmal manifestations we have a valuable index. Reduction in total solids, especially in urea and phosphates, with the inverted proportion of alkaline and earthy phosphates is, perhaps, only found in hysteria.

Once the suspicion of hysteria is entertained, a painstaking examination of the patient should confirm or banish it. The greatest danger arises from failing to appreciate the limitations of hysteria and allowing its presence to interrupt careful search for organic disease of which it may be a secondary expression. Too often, when hysteria is recognized, the physician is content to attribute every symptom and complaint to the psychoneurosis. Hysterics may have pituitæmia, Bright's disease,



cerebral hemorrhage, typhoid fever, and lip-disease, as well as other persons. In every hysterical case the individual must not be forgotten or underlying disease overlooked.

**Treatment.**—Recognizing in hysteria a mental disturbance principally, the treatment must be mainly psychic. This point of view may at once strengthen and weaken the physician. Unless he has a clear conception of the power of mental therapeutics, he is likely to look upon all measures as mere placebos and to look faith in their value. Not confident himself, he fails to inspire confidence in the hysterical patient. The game is lost before it is begun. It is the self-confidence of the charlatan or the fanatical belief of the "Christian Scientist" that now sometimes succeeds when well-informed physicians fail. Expectant attention and hopeful anticipation have cured hysteria in all ages, and are potent measures to-day. Methods are usually successful in proportion as they are novel to the patient, strike the fancy, and stimulate the imagination. This serves in itself to distract the hysteric from the rut of his fixed ideas, and, if the assurance of help and cure is added and constantly repeated by suggestion, it tends to supplant the morbid condition. A great moral impression or a mental shock may terminate hysteria at once or may greatly aggravate it. Treatment may be considered (1) as *general*, applicable to all manifestations of the disease; and (2) *special*, regarding the manifestations of individual cases.

**General Treatment.**—The first consideration is, if possible, to decipher the *fixed idea* that dominates the patient. This is easily done when the hysterical syndrome originates in some serious mental storm or personal experience. In other cases it can be inferred from the hysterical manifestations, and occasionally it is constantly expressed in some worry or apprehension. In many cases, however, it is a subconscious idea. It may have arisen even in a dream, or it may be so intangible that it never is fully formulated in the patient's consciousness. Sometimes, from motives of shame, or modesty, or morbid conscientiousness, it is studiously concealed. The so-called psycho-analysis of Freud concerns itself especially or solely with this view of hysteria, and in an endless search for some sexual item rarely fails to find it.

When once the end of the tangled skein is in the physician's hands, his task is to modify or *destroy the fixed idea*, and thus remove the source of morbid mentalization. Too often family and friends support the patient's morbid view and exaggerate the gloomy prospects, adding fuel to the flames by anxious solicitude and thinly veiled or openly expressed fears. In the highly suggestible condition of hysteria their constant presence and their consciously or unconsciously reiterated depressing suggestions counteract all possible good at the hands of the physician. The very locality in which the disease has developed constitutes a forcible reminder of its present and prospective woes. Unless the surroundings, companions, and vicarities of a hysteric can be absolutely controlled, it is usually impossible to manage the patient. It is for this reason that *isolation and separation* from everything associated with the patient's morbid past is usually the first and most essential requirement of treatment. Under new circumstances the statements of the physician regarding the trivial nature of

the dominant idea or his orders to dismiss it and his assurance of cure carry a weight and force that are not immediately destroyed by other more constant and less wholesome influences. The very fact of isolation is a profound influence that can readily be guided into a hopeful and helpful channel. A visit from an anxious mother or solicitous friend may, in a few minutes, destroy all advantage and recall the morbid past with added intensity. This plan of treatment can often be put in the form of the Weir Mitchell rest-cure, and requires the same conditions already indicated in the treatment of neurasthenia (p. 608). Mild cases, especially in the young, can sometimes be well managed by a long journey with a sensible and not too sympathetic companion, or by a protracted visit to friends or relatives properly informed of the attitude they should maintain toward the patient. The so-called "shell shock" cases occurring in military practice in the World's War have abundantly proved all former experience, and the readiness with which many such cases have been cured by persuasion, often coupled with intensive painful electrical applications, constitute nothing new except the wholesale scale in which these cases have been encountered.

*Hypnotism* in its concentrated form is a dangerous measure and only of occasional service. In the hypnotic state the patient may readily disclose the hidden or subconscious idea, and it may at once be attacked and destroyed by counter-suggestion. In the same way progressive improvement or immediate relief from the various conditions present in the patient may be suggested. Hypnotism may, however, precipitate a latent hysteria, and patients hypnotized for the removal of trivial hysterical symptoms have, in the hypnotic state or immediately after it, blossomed out in all the manifestations of major attacks or developed protracted paralysis and contractures. In other cases hysteria restrained by hypnotism has recurred with added force when the sittings were discontinued, and Féré goes so far as to consider hypnotism but a transformation of hysteria. It should be held as a last resort.

It goes without saying that anemia and general states call for such remedies as are ordinarily beneficial, and local disease presents exactly the same indications as in non-hysterical patients.

**Special Treatment.**—The *convulsive attacks* can frequently be stopped by a dash of cold water, by a sharp command, by pressure on hysterogenic zones, and, if other means fail, by inhalations of ether. Their repetition depends upon conditions which must be met by the general measures previously indicated. *Paralyses and contractures* are among the most permanent accidents when once established, and become actual stigmata. If taken early, they can usually be managed. Massage, electricity, and repeated assurances of their early cure and of their insignificant importance is usually sufficient if the friends, in the way already described, do not defeat these measures. After a long duration they may require the full isolation treatment. *Contractures of years' standing* may be followed by fibrinous contractions only amenable to surgery. *Anesthesias and hyperesthesias* can be modified by a number of influences of the esthesogenic sort. Faradic and Franklinic electricity often act very readily to reduce and completely disorganize the field of disturbed sensation. Various objects, metallic, wooden, etc., active or inert magnets, have the same influence. It is all a matter of



concentrating the fixed and hopefully expectant attention upon the parts. In the same way these measures are useful in the palsies and contractures. *Spinal irritation*, so called, when once well marked, usually requires the isolation and rest treatment, but sometimes the actual cautery or flying blisters, or other heroic and hence impressive measures, succeed in removing the hypersensitiveness. *Aphonia* and *mutism* require treatment of a similar sort. A faradic electrode introduced into the larynx or pharyngeal cauterization has succeeded almost instantly in some cases, but lasting benefit usually follows persistent and repeated suggestion of steady improvement, coupled with various suggestive manipulations of the parts. *Laryngeal spasm* and hysterical cough or sneezing, or diaphragmatic spasm, generally can be interrupted by having the patient or a nurse forcibly pull on the protruded tongue. *Laryngeal spasm* in rare instances may require anesthesia or even tracheotomy, but, fortunately, spasm is commonly followed by relaxation of the spasm. *Dysphagia* from *oesophageal spasm* is often relieved by passing the stomach-tube and demonstrating the permeability of the gullet. *Vomiting* is sometimes benefited by lavage of the stomach and the mechanical introduction of food, but assurance of improvement and suggestion must be added to all these measures.

Finally, it is the physician who is most sure of himself and of his diagnosis and has a distinct idea of the mental side of hysteria who best succeeds with general or special treatment.

## CHAPTER XL.

### EPILEPSY.

EPILEPSY has been the subject of medical description since the earliest times. It was known to the ancients as the sacred disease, *morbus sacer*. In colloquial English it is called the "falling sickness." Its most characteristic manifestation shows forth in the derivation of the name "epilepsy," which implies being seized upon. The seizing has been variously attributed to mythological deities, to the possession of devils, and to vapors and humors arising in the body. Focal epilepsy, or Jacksonian epilepsy, resulting from cerebral injury or disease, may be omitted from the present consideration. Epileptical attacks arising from alcohol, lead, and uremia, and the eclampsia of parturients have no necessary relation to the epilepsy now in question. Epilepsy can scarcely be considered a distinct disease. It is a syndrome of nervous and mental symptoms appearing under a variety of pathological states. In many instances it is associated with morphological cellular changes in the cortex. In many more cases the anatomical basis still escapes detection. So commonly is epilepsy presented by neuropathic and psychopathic strains, and in those physically or mentally defective, that in itself it may be considered a presumptive indication of degeneracy. It is statistically proved that there is more than one epileptic for every four hundred of the population in this country.

**Etiology.**—*Heredity* plays a very important part in the causation of epilepsy. Arthritis, syphilis, phthisis, inebriety, insanity, and neuroses are common in the antecedents. Epilepsy appears frequently in succeeding generations, and may descend directly from parents to children, but is more likely to be indirectly propagated by way of collateral branches. The heredity is most often by transformation from other neuropsychic disease. Thus, hysteria, epilepsy, and idiocy may follow in successive generations. Epilepsy among cousins is more frequent than among brothers and sisters, where, however, various neuropathic equivalents are frequently encountered. *Concognition* plays no part unless it brings together individuals of similar nervous or mental defect.

Epilepsy may appear at any age, but it is distinctly uncommon for it to commence after the age of thirty. Epileptoid attacks after that age should always awaken a suspicion of gross organic brain, heart, or kidney disease. Syphilis is the usual cause of such seizures from thirty to forty-five; after forty-five we encounter the degenerations of senility, vascular changes, and accidents. The very great majority of cases of epilepsy develop under twenty years of age, and the pubescent period, between twelve and seventeen, contains the greater proportion of them. Very frequently epilepsy begins in infancy. Munson<sup>1</sup> publishes a chart based on 2733 cases admitted to the Craig Colony, which shows the disease to have appeared within the first year of life in 22%, the largest number in any one year. The incidence then rapidly falls to the tenth year, when it again mounts throughout the adolescent period, gradually descending after twenty. Convulsions during the first and second dentitions, incited by any febrile, septic, or toxic cause, may be followed by epileptic attacks at puberty. At least such attacks must be looked upon as demonstrating a spasmophilic constitution. In some cases, beginning as eruptive attacks in infancy or early childhood, epilepsy follows, with more or less periodical attacks from that time. Some families present numerous deaths from infantile convulsions, and epilepsy sometimes develops in those who escape. Nocturnal enuresis, pavor nocturnus, and epilepsy may appear in the same case, apparently replacing one another. It is necessary to carefully exclude from this consideration that large number of cases in which cerebral injury is present or brain defect arising from intra-uterine, birth, or post-natal causes. Such instances usually present evidence of cerebral lesions in some form of palsy, and are considered in Part III. Males are somewhat more commonly affected than females, with a ratio of 20 to 16, according to Spearling. Munson<sup>2</sup> conclusively shows that the life expectancy is greatly reduced in epilepsy, the average duration of the disease in 555 cases being 17.58 years. Lung diseases, including pneumonia, phthisis, and pulmonary edema, are the most common causes of death, followed by status epilepticus, series of attacks, and accidents occurring during fits, or states of epileptic automatism. No doubt the severe pulmonary congestion present during severe attacks induces pulmonary susceptibilities.

**Inciting Causes.**—The alleged inciting causes of epilepsy, by

<sup>1</sup> Med. Record, January 8, 1901.

<sup>2</sup> Loc. cit.



their number and relative harmlessness, reinforce the presumption of a necessary predisposing defect, in the field of which they may become active. The hereditary considerations, the usual evidence of degeneracy, and the common-onset of the disease at developmental epochs all speak of a vicious organization unequal to the shocks of ordinary life and the demands of growth. In some cases, however, it is impossible to fix upon any predisposing state.

Toxic agents, such as alcohol, lead, mercury, tobacco, chloroform, ether, morphia, cocaine, etc., have been accused of inciting epilepsy. Alcohol is certainly competent to do so. The first fit may follow a drinking bout. Other *toxic conditions* arising from the intestinal tract or due to defective elimination through the kidneys may act as inciting causes. The *infectious diseases*, such as the exanthemata, may furnish the starting-point for epilepsy. *Typhoid fever* is causative in some instances. *Syphilis* may be causally related to epilepsy in various ways: (1) As a hereditary factor; (2) through the malnutrition of the secondary period; (3) by local cerebral disease; and (4) Fournier thinks there is a peculiar syphilitic variety of epilepsy appearing late in the lactic history. *Head injuries* may produce focal epilepsy, but sometimes set up the common form of the disease. In the Franco-Prussian War, as a result of 8385 non-fatal head injuries, 46 cases of epilepsy developed, only about  $\frac{1}{2}$  of 1 per cent. All things considered, head injury must be considered as a rare cause of epilepsy. *Reflex irritations* due to nasal growths, intestinal worms, pelvic disease, genital abnormality or disease, masturbation, and eye-strain are occasionally sufficient to provoke epileptic attacks and to cause their repetition. Sensitive scars, decayed teeth, and ingrowing toe-nails have also been brought into this list. In fact, any local peripheral cause of constant nerve-fag may, in those of unstable organization, serve to upset the nervous apparatus. Physiological states, like menstruation and childbirth, may incite epileptic attacks in those predisposed. In female epileptics, the menstrual periods are very frequently the occasion of attacks. *Mental shocks*, particularly fright, may cause the first fit. Some epileptics have a fit upon the incidence of any emotional disturbance.

**Pathology.**—It would be impossible within reasonable limits to give the numerous theories that have been advanced regarding the nature of epilepsy, or to discuss at length the various locations assigned the morbid action resulting in the fits. Suffice it to say that the presence of mental symptoms, sometimes the limitation of the attack to unconsciousness alone, the experimental demonstration of cortical excitability, the usual organic basis of Jacksonian fits, the recent findings of changed cortical structures in some cases, and a recognition of the supremacy of the cell in the doctrine of the neuron theory, all combine to declare epilepsy a disease of the cerebral cortex.

It must be admitted that at present the most careful search fails in many cases to detect any histological changes known to be morbid. This is only another way of stating our ignorance of certain pathological processes and the limitations of present methods of research. When a patient dies as the result of repeated convulsions, venous and sinus engorgement, minute hemorrhages, and more or less extensive

cerebral ecchymoses merely evidence the recent paroxysms. Lesions of longer standing have been frequently found, such as meningo-encephalitis, widely disseminated tuberculous sclerosis in the cortex and the great ganglia, and diffuse sclerosis of the convolutions. Much importance was once attached to such hypertrophic sclerosis affecting the cornu ammonis or appearing in the bulb. Chaslin, Féré, and many others have repeatedly found neuroglial proliferation. Bloch and Marinsek have described vascular and perivascular changes in the cortex. Van Gieson has demonstrated changes in the large pyramidal cells of the cortex and in the neuroglia in two cases in which portions of the cortex were removed by operation. Bleser,<sup>1</sup> in twenty-six epileptic brains, found a definite, wide-spread hypertrophy of the neuroglial bundles lying between the pia and outermost nerve-bundles. This change was not proportional to the severity or duration of the disease or relative to the mental degradation of the patients. In fifty-four non-epileptic brains similar changes were not found. Olschneker<sup>2</sup> draws prominent attention to persistent thymus and enlarged intestinal glands. Clark and Pratt<sup>3</sup> describe wide-spread changes in the cortical cells, especially of the second layer, consisting of deformities, absence of chromatic substance, and swelling of the nuclei with tendency to loss of the nucleus under the section knife. Denath<sup>4</sup> shows that cholin is usually present in the blood, which, with other products of metabolism, such as uricemia and kreatinin, may act to bring about the attack. The blood shows an excess of eosinophilic cells which diminishes in the three days preceding an attack<sup>5</sup> and returns to its maximum within ten hours after the attack, often being of subnormal number during the attack. Turner<sup>6</sup> lays stress upon the apparent lack of full development of many cortical cells and the persistence of subcortical cells indicating imperfect differentiation and growth. Additionally he describes groups of shrunken cells and acute cellular changes similar to those produced by ligating cerebral arteries in animal experiments. On the part of the blood he notes an excess of blood platelets and various forms of intravascular clotting and cortical hemorrhages.

**Symptoms.**—The most striking symptoms of epilepsy are the "attacks" or fits and their modifications and equivalents. Of equal importance are the postepileptic states and the mental and physical conditions of epileptics.

**Epileptic Attacks.**—Prodromes and Auræ.—Many, but by no means all, epileptics have some distant or immediate premonition of the attack. Friends, nurses, and attendants upon epileptics often learn to anticipate an attack by temperamental or facial changes that are unnoticed by the patients themselves. These may be present several hours, or even several days, before the convulsive explosion. Forerunners of a motor sort are grinding of the teeth in sleep, twitching of the eyelids, face, or extremities, tremors, and restlessness. Some epi-

<sup>1</sup> "Munch. med. Wochensh.," No. 33, 1885.

<sup>2</sup> "Zell. u. Gew. Hesp. für Epileptics," 1886.

<sup>3</sup> "Boston Med. and Surg. Jour.," April 21, 1901.    <sup>4</sup> "Med. News," June 28, 1895.

<sup>5</sup> Minelli and Pissare, "Riv. Sperim. di Frenet.," 1906.

<sup>6</sup> "Ec. Med. Jour.," March 3, 1906.



leptics complain of tingling in the extremities or on the surface of the body; others have peculiar sensations on the tongue or in the nose, a tickling of the palate, or disturbances of smell or taste. Others have photophobia or brilliant visions. Ringing in the ears, genital excitement, respiratory oppression, vague discomfort, mental stimulation or depression, unusual irritability or gaiety, great increase or decrease of appetite for food, are some of the various premonitory conditions. Some patients look unusually pale; others present a suffused, puffy countenance; and temporary erythematous and urticarial eruptions have been seen. Much as these more or less prolonged premonitory conditions vary among themselves, they are comparatively uniform and constant for a given case.

The *aura* is the particular motor, sensory, or psychic feature that immediately announces the attack, and may be considered its initial symptom or phase. In some instances it represents an abridged attack. It may recur for years unrecognized, until a fully developed fit indicates its true character. An *aura* of some sort occurs in fully one-half of all cases of epilepsy. *Motor aura* may consist in a sudden limited muscular contraction or tremor, similar to the signal symptom of Jacksonian fits, and of only momentary duration. Automatic movement, and even a series of coördinate acts, may introduce the generalized attack. A patient scratches at the pit of the stomach, or winks his eyes, or moistens his lips with his tongue, or makes a few swallowing efforts, starts forward or backward, rotates upon his vertical axis, coughs spasmodically, springs up, or rolls his eyes to one side, following with his head and body. Unconsciousness at once follows, and the full fit occurs. Many epileptics have *sensory aura*. A peculiar sensation is described, commencing in the feet or anywhere in the extremities, or in the abdomen, especially at the pit of the stomach. It mounts upward, and consciousness usually fails as it reaches the upper part of the chest or the neck and head. In some instances it strongly suggests the globs of hysteria. One patient describes it as a "ragging feeling," and it usually is of an indefinite and indescribable sort. Visual and auditory sensations are the most frequent of those that relate to the special senses. Those of taste and smell are very rare. Uniform and persistent aura of taste and smell are so commonly symptomatic of temporal lobe disease, and particularly of the uncinate gyrus, that convulsions so introduced are frequently called *sacinate fits*. Some epileptics hear certain sounds of a pleasant or startling sort; sometimes it is a distinct voice. Others describe everything as turning black or dark before their eyes; some have phosphores or bright flashes; and in exceptional instances a definite picture is presented, such as that of persons or animals, which may rapidly approach or recede.

Jackson first called particular attention to the *psychic aura*, which are as vague and varied as those of a motor and sensory character. A sudden recollection or the instantaneous review of a lifetime, a certain fixed idea or doubt, may announce the convulsion. Sometimes it is a rapidly growing mental depression, a fervor, a feeling of mental exaltation or weakness, irritable impulses, gaiety, rage, or quarrelsomeness,

Another group of auras are called *visceral*, but are, for the most part, sensory or motor manifestations referred to the various organs. Pre-cordial pain, violent cardiac palpitations, respiratory anguish, laryngeal spasm, weight at the pit of the stomach, vomiting, colics, and sudden imperative demands to evacuate the bladder or bowels are the principal ones. *Cerebral* auras, such as sudden pain in the head, vertigo, stammering, and paraphasia, have been noted.

Leclerc has noted an elevation of body temperature during the aura, and Valsin has observed increased temperature in the limbs in which the aura originated. Féré and Franck have noted an increase of arterial tension before the fit, and the writer has obtained a sphygmogram showing the same thing. All these considerations indicate that the aura is of cerebral origin.

Epileptic fits vary greatly in different cases, and usually a patient presenting severe convulsions also has slight or abortive attacks. We may consider (1) the complete attack, (2) the incomplete attacks, and (3) the equivalents of epileptic attacks.

The complete convulsion, preceded by an aura or not, comes on suddenly, consciousness and sensibility are instantly lost, and the patient falls. The fit presents three distinct periods: (1) one of tonic convulsion; (2) one of clonic convulsions; and (3) a period of stertor.

The tonic stage opens suddenly, with all the muscles of the body in tetanic rigidity. It results that the patient not only falls, but is frequently violently thrown down. At the same time, the sudden tetanic muscular grasp of the thoracic cage and the fixation of the laryngeal apparatus give rise to a cry that is practically never varied. Sometimes it is a shriek; more often it is an expiratory guttural noise. The face is at first pale, but rapidly becomes congested and cyanotic, because respiration is inhibited. The eyes are fixed, often turned up or to one side or convergent, and the pupils are rigidly dilated. The dilatation is proportionate to the severity of the attack. Contracted pupils may sometimes be seen at the onset of the attack and again at the beginning of the stage of stertor. Minute vascular ruptures in the skin, mucous membrane, and conjunctiva often occur. It is probable that similar vascular accidents take place in the internal viscera and in the brain. If the hand be placed on the tonically contracted muscles, a thrill or vibratory impulse will be detected, such as is occasioned by excessive voluntary efforts. The tonic period is one of apnea, and only lasts one to two minutes at most. It may last only a few seconds. The tongue is frequently thrust between the teeth, and may be lacerated. The head may be slightly turned or retracted, the trunk is rigid and usually straight, the upper extremities, flexed at the elbows, are held close to the trunk, the fists are clenched, or the fingers may be made into a cone or spread by the interossei. The lower extremities are constantly rigidly extended. Complete symmetry of movements and position of the limbs is exceptional and quite frequently the lower limbs are partially flexed. It is at this stage usually that the spasm of the abdominal muscles expels the contents of the bladder, sometimes those of the rectum. The vascular strain is extreme.



The *clonic period* is in some sense a continuation of the tonic stage. The muscles relax, and again suddenly contract. The limbs, body, face, and all movable parts are agitated by the sudden sharp-twistings, which cause the face to grimace horribly, the jaws to grind together, often lacerating the tongue and cheeks, the eyes to jerk in their sockets, the air to be noisily forced in and out of the chest, churning the saliva in the mouth and throat and forcing it through the teeth and lips in a bloody foam. The arms, legs, and head beat against the ground. The clonic movements, at first rapidly repeated, gradually become less frequent, but maintain their force and vigor to the very last movement. The respiration, dominated by the clonic spasm, is inefficient; the asphyxia and cyanosis persist; the cardiac movements are precipitate; the arterial tension is intense. This stage lasts from one to five minutes, and terminates in full relaxation, in which the bladder and rectum, if not emptied in the tonic period, may allow their contents to escape. The body and face are covered with perspiration, the temperature mounts two or three degrees Fahrenheit. The clonic movements may be bilaterally synchronous, but have no suggestion of purpose, and are unmethodical and incoördinate.

*The Period of Stator.*—The relaxation in which the clonic stage ends is usually marked by deep stertorous breathing and continued unconsciousness. The patient lies inert, just as the convulsion left him, profoundly comatose, and may perish at this time, smothered by the bed-clothes or by similar misdeeds. After a few minutes, perhaps after a half hour or more, he opens his eyes and regains partial control of himself, without any knowledge of what has happened during the attack. If the attack take place during the night, the stertorous period may gradually merge into natural sleep, and the patient may be entirely unconscious of the occurrence of the fit. Usually the fit leaves the patient more or less confused, fatigued, sore, strained, and subdued for several hours or for a day or two.

While the typical fit presents all of these symptoms, it may be infinitely modified. The tonic period may be instantaneous only. The clonic movements may be confined to the face, upper extremities, or lower limbs. The cry, urination, stertor, or bitten tongue may any or all be lacking. The convulsion may be most marked on one side, and, indeed, exact symmetry is uncommon. The only constant feature is disturbed consciousness, but unconsciousness does not in itself constitute a fit, though it may practically constitute an equivalent.

Epileptic attacks often occur during the night, and in some instances only during sleep, giving rise to the clinical variety called *nocturnal galegas*, which may last for years without being suspected. Fits are likely to occur just as the patient is getting to sleep, or just as he is awakening in the morning. They may be induced by emotion, especially fright. Fatigue, coitus, menstruation, and child-birth may provoke them. Treppe<sup>1</sup> notes that the last days of the menstrual flow and the two or three succeeding days are those on which the fits are most

<sup>1</sup>"L'Encéphale," 1908, No. 6.

likely to occur, and that the menstrual periods are commonly marked in epilepsy by mental stimulation, or depression with irritability. In most cases, however, convulsions take place without adequate traceable cause. Some patients have them very frequently, and with considerable regularity; others have them at irregular intervals; and others may have but two or three during a lifetime. In aggravated instances they occur in groups, several taking place within a day. Such groups frequently follow an interval more than usually prolonged, or succeed a period of brouid repression.

Occasionally, the patient, without recovering consciousness, has fit after fit, and in this way a series of hundreds may occur. This constitutes the *status epilepticus*, the gravest form of epileptic paroxysm. It consists of (1) a period of incessant convulsions, and (2) of a period of prostration and collapse; but sometimes death occurs in the convulsive stage. Usually, after the fits have continued for several hours, or even for several days, they become less severe, and finally cease, and collapse ensues. During the convulsions the respiration and pulse are accelerated and the temperature may reach  $104^{\circ}$  or  $105^{\circ}$ , and even rise higher, until death occurs. The individual fits of the status epilepticus are rarely of the severest variety. The stupor is penetrated more or less regularly by convulsive attacks of moderate force, and the status may even be made up of the limited attacks of the incomplete variety, such as vertigo, delirium, stupor, coma, cough, hiccup, and various psychic states. The collapse of epileptic status may be so profound as to reach a fatal ending.

**Incomplete Attacks.—Petit Mal.**—Fractional epileptic attacks present an endless variety, of which only a few of the common forms can be indicated. Almost any part of the full attack as previously described may alone constitute the limited seizure. Very frequently the disease presents as aura and the attack aborts. This may occur repeatedly, even for years, before the major seizures develop. Such warnings may occur in the intervals between the full attacks or after they cease, if the disease subside spontaneously or under treatment. A large number of cases present vertiginous attacks. The patient suddenly loses consciousness and falls, or nearly falls. There is usually slight twitching of the face, or perhaps only a little quivering about the eyes or mouth, or a deviation of the head and eyes, which may pass unperceived. Almost instantly the patient recovers himself, but may feel weak and faint. In other instances the patient falls heavily, but clonic movements and stertor do not develop. Again, the patient may fall, and become at once comatose and stertorous, as in apoplexy.

Of great interest are the attacks marked mainly by a momentary loss or disturbance of consciousness. The face may suddenly pale and the eyes look vacant. Whatever is being done is interrupted, and immediately resumed. The patient ordinarily has no knowledge of anything unusual having occurred. In talking he suddenly stops, looks absent-minded, and takes up the sentence where it was broken. A musician may pause one or two bars, and continue. At table the cup is poised



half way to the mouth, etc. In other instances a little twitching of the face is noticed, or the patient drops or throws whatever he has in his hand, and usually there is momentary rigidity during the brief unconsciousness. In some instances a few words are muttered, or a little saliva dribbles from the mouth, and in such attacks the escape of urine is far from infrequent.

In rare instances the patient, under an irresistible impulse, rapidly walks or violently runs for a few moments in an automatic, unconscious way, and may fall finally in a fit, or may recover consciousness without an intervening convulsion; or after a fit a patient may suddenly run for some distance (*epileptic procession*). Attacks of stupor or profound sleep have been recorded, into which the patient falls at intervals, and during which he can not be aroused. These may alternate with convulsive attacks or may be replaced by them. Attacks of generalized or localized trembling, local spasm, and salivary spasms may constitute the epileptic paroxysm. As before said, the only constant feature in epileptic attacks is a disturbance of consciousness during the period of the attack. Cui<sup>1</sup> has observed sudden falling of body-temperature for about an hour, amounting to several degrees, sometimes recurring several times a day, and apparently constituting an epileptic equivalent.

**Psychic Equivalents of the Epileptic Attack.**—Instead of epileptic attacks of the ordinary kind, or in alternation with such, or as a repeated prelude to the major convulsions, or immediately following the fits, we may have a variety of acute mental disturbances. Epileptics may unconsciously, automatically, and with apparent purpose perform a number of coordinate acts. Homicidal, suicidal, or pyromaniac acts may thus be done by epileptics, or intricate maneuvers, such as require the use of tools, may be accomplished. Subsequently, as a rule, they have no knowledge of such acts. The precursive fit may, in a sort of status, be prolonged so that the patient may make long flights, or, in a less violent way, make journeys of several hours' or several days' duration, during which the conduct is so natural as to attract no notice. Self-consciousness usually is rather abruptly restored, and they are astonished to find themselves at a distance from home, with an intervening blank period of time. Sudden wild, manual outbursts, in which the patient may be destructive and dangerous to others, are encountered, and these may terminate suddenly or be protracted for several days, attended by great excitement, a high pulse and temperature, and subsequent collapse. Sometimes such attacks are stopped by a fit, or they may succeed a severe convulsion, or they may take the place of the convulsion. Epileptic automatism may, in a static form, last days and weeks, during which the patient conforms naturally to his surroundings, but subsequently has little or no knowledge of the automatic period.

**Postparoxysmal Phenomena.**—As physicians in practice rarely have the opportunity of seeing the patients in epileptic attacks, a knowledge of the postparoxysmal phenomena is important. As a

<sup>1</sup> "Gazzetta. I. Nervosa, u. Psichiat.," Oct., 1900.

result of the cortical exhaustion, a number of transitory symptoms follow the usual seizure. Tremor, paresis, disturbances of speech, of general sensibility, and of the special senses have sometimes been observed; the muscular strength is commonly reduced, the knee-jerks are diminished or abolished, and muscular tone is lessened. Omsl,<sup>1</sup> however, insists that during the convulsion and in the postepileptic period the reflexes are as commonly increased as diminished. If the attack is severe the tendency is to exhaustion of the reflexes. Slight attacks are likely to be followed by some increase of reflex activity. Babinski's toe-sign is usually present on both sides during the period of stertor and in the comatose states that sometimes follow or substitute the attacks, and may persist for some hours. The pupils tend to continued dilatation.

Several attacks, repeated within a few hours, lead to an appreciable loss of body weight. The urine, even after a single attack, shows an increase of phosphates, particularly of the earthy phosphates, and of the nitrogenous constituents. The temperature is commonly subnormal. This may occur even in petit mal and has been noted in the brain itself by Miris.<sup>2</sup> The toxicity and acidity of the urine are increased, and Agostini<sup>3</sup> finds the gastric juice has toxic properties. These properties are most marked just before and after a fit, and are proportionate to the intensity of the attack. Vomiting after an attack is not uncommon and almost invariably causes the fit to be attributed to errors of diet, which is sometimes the case. Calotto<sup>4</sup> claims that the sweat of epileptics is hypertoxic proximate to the fits, and urges the use of hot baths in the treatment of the disease, asserting good results from their employment. The special toxicity of the excretions in epilepsy is not proven. The arterial pressure is reduced, crises of polyuria, diarrhea, sweating, and salivation may be encountered. The presence of petechial ecchymoses, the bitten tongue, the voided urine, or fecal discharge is to be noted. Ordinarily, there is mental hebetude and sluggishness, a tendency to languor, and a desire to sleep. Headache is usually complained of, and often there is a feeling of extreme muscular fatigue and soreness. The psychic depression may reach a stuporous degree, and this may follow attacks that lack muscular violence. There is a decided tendency to defective memory and mental deterioration, which, in the majority of cases, presenting frequently repeated major or minor attacks, markedly tends to dementia in the end.

**Myoclonus Epilepsy.**—In rare instances myoclonia and epilepsy occur in the same patient, constituting myoclonic epilepsy or the "association disease." Such cases usually show marked degeneracy, and the disease begins early in life. Epilepsy usually antedates the myoclonic features which when once developed usually increase in severity and commonly culminate in epileptic convulsions.

The myoclonus of the association disease is somewhat atypical compared with the essential myoclonias.<sup>5</sup> The contractions are usually lightning-like, but may have a fibrillary character, involving certain

<sup>1</sup> *Am. Med.*, Jan. 30, 1903.

<sup>2</sup> *Annali di Neurologia*, 1899.

<sup>3</sup> *Riv. di Pat. Nerv. e Ment.*, ix, 1896.

<sup>4</sup> *Riv. Spec. di Frenal.*, 1897.

<sup>5</sup> Clark and Prout, *Am. Jour. of Insanity*, Oct., 1902.



parts of muscles only,—a condition described as “live flesh,”<sup>11</sup>—and such manifestations are likely to develop into typical myoclonic contractions, though they may remain fibrillary through the life of the patient. A single general tonic contraction may rarely constitute the entire clinical picture. Myoclonus contractions end imperceptibly in the tonic stage of the epileptic paroxysm. The contractions are often strong and affect large masses of muscles, rendering locomotion difficult. The trunk is frequently affected, causing the body to jerk backward and forward and laterally. The face and distal portion of the extremities are frequently involved. Generally the myoclonus is symmetrical, but both sides are not always synchronously involved. Commonly it begins in the upper extremities, and in a few days or weeks involves the lower extremities, the body, neck, and face in the order given, the muscles about the eyes and mouth being the last affected. The tongue and diaphragm frequently suffer, in severe cases producing grunts and barks. As the malady develops the myoclonus becomes more and more persistent during the waking state, having a tendency to develop the myoclonic status, which may terminate in death. As a general rule, superficial and deep reflexes are increased, and also, as a rule, there is lack of physical and mental development.

The prognosis as to recovery is extremely poor. Those who have reached mature years present extreme senility and progressive dementia.

The diagnosis in a typical case is easy. There are family types and sporadic cases, the majority being of the latter variety. The condition may be acute and severe or mild and chronic.

The treatment can be only palliative, but, as a general rule, long remissions both in the myoclonus and the epileptic course may be obtained by the use of bromids.

**Continuous Epilepsy.**—Under the name of *epilepsia partialis continua*, several Russian writers, notably Koshevnikov, have described a rare form of epilepsy, marked by characteristic major attacks, but also presenting in the intervals persistent clonic movements of limited extent. These clonic movements are sometimes of such vigour as to require the constant use of the unaffected hand to steady them. Their gradual increase in severity leads to a full attack. In some instances they have persisted during sleep. A somewhat similar fractional status is also at times observed in Jacksonian cases and in organic brain lesions.

**The General State.**—The bodily health in epileptics may leave little room for complaint. Very commonly they present gastric disturbances, a sluggish skin, and constipated bowels; but usually this is due to the bromids with which these patients are so commonly saturated. Many epileptics have generalizing habits and insatiable appetites for food. This, with the inactivity often forced upon them by the disease and reinforced by the bromids, leads to fatty fitness. As a rule, they are indifferent and careless, and often the finer mental attributes and the keener sensibilities are dulled early in the disease.

**Diagnosis.**—The diagnosis of epilepsy is often very difficult, if the attacks are incomplete or not open to intelligent observation. The

disease in the nocturnal form, or in the varieties of *petit mal* with slight attacks, may not attract attention for years. When the suspicion of epilepsy is aroused,—as, for instance, by bed-wetting, bloody stains upon the pillow, unexplained bruises, conjunctival ecchymoses, a dislocation or fracture occurring during sleep, cuts on the face or scalp, a bitten tongue, or some automatic or convulsive attack,—a careful investigation will rarely fail to disclose the nature of the disorder. In such cases a history of repeated momentary unconsciousness, or of some of the various sensory auras, is significant. An account of nocturnal incontinence or nocturnal pavor in childhood is suggestive. Convulsions during teething, the marks of degeneracy, or the presence of a family neuropathic taint have some weight. If there have been repeated convulsions, and they conform to the epileptic type, presenting sudden onset, with or without warning, tonic, clonic, and stertorous stages, and complete recovery, there is little room for doubt.

We have, however, to exclude the epileptoid fits symptomatic of various functional and toxic conditions. Intestinal, renal, metallic, drug, and alcoholic poisonings may cause convulsions that very closely, or exactly, copy the attacks of epilepsy, and they may induce epilepsy in those predisposed. Vertigo of gastric, neural, ocular, and neuro-themic origin may suggest *petit mal*. *Hysterical convulsions* are often mistaken for those of epilepsy. *Oxygenic brain disease*, such as tumors, cerebral palsies in children, and brain injuries, may occasion epileptiform attacks. Finally, epileptic seizures may be of the apoplectic form and suggest *cerebral hemorrhage*, or the psychic equivalents may be confused with *transitory monie*. In every case it is obligatory to examine the patient rigorously from head to foot, both for the purposes of diagnosis and treatment. Of epilepsy, there is no one pathognomonic symptom. The following table shows some of the differential characters of epileptic and hysterical convulsions:

TABLE OF DIFFERENTIAL CHARACTERS OF EPILEPTIC AND HYSTERICAL ATTACKS.

THE EPI.	EPILEPTIC.	HYSTERICAL.
Prodromes.	Mental or physical perturbations.	Emotional disturbances.
Aura.	Common, but momentary.	Common and of considerable duration.
Onset.	Sudden; complete; cry, fall, rigidity.	Gradual.
Consciousness.	Instantly lost.	Partially lost or retained.
Course of convulsion.	Tonic, clonic, and stertorous stages.	Epileptoid and emotional phases.
Duration.	Two to five minutes.	A few minutes to several hours.
Positions.	Governed by flexors, mainly.	Tendency to extension, <i>arc de cercle</i> , episthomas, cruciform attitude, etc.



TABLE OF DIFFERENTIAL CHARACTERS OF EPILEPTIC AND HYSTERICAL ATTACKS  
(Continued).

THE FIT.	EPILEPTIC.	HYSTERICAL.
Eyes.	Pupils dilated and rigid.	Pupils generally mobile and active.
Tongue.	Usually bitten.	Bitten very exceptionally.
Mouth.	Frothing common.	Frothing absent.
Sphincters.	Relaxed, usually.	Usually contract.
Pulse.	Accelerated greatly and tension increased.	Rate and tension not much changed.
Temperature.	Elevated 1° or 2° F.	Normal.
Termination.	In hysterical, gradual.	Rather prompt ending and little discomfort.

**Prognosis.**—As an exception, but proving the rule of gravity in the prognosis of epilepsy, rare cases recover spontaneously. In a general way there is little hope for cure, if the disease, with repeated attacks, has lasted over two years, or been intense in its manifestations for even a shorter period. There is some tendency for it to appear, subside, reappear, or increase at the developmental epochs of dentition, puberty, adolescence, and the climacteric. Any inciting cause that is controllable is a favorable consideration. Epilepsy as a manifestation of degeneracy is of bad import. If there is any reason to suppose that organic cortical changes have occurred, or if dementia has appeared, the outlook is unfavorable. Grand mal cases offer a slightly better prognosis than petit mal or psychic cases. In those of a distinctly spasmodic tendency or a surcharged neurotic heredity the prognosis is rather more favorable than in the absence of such factors. These individuals readily develop epileptic attacks, often as the result of remediable conditions, which in those of a more stable organization are of insignificant import.

A single attack is almost never fatal of itself, but a condition of status is very likely to terminate fatally, and every attack undoubtedly leaves some harmful trace. Rarely a severe attack owing to some cardiac weakness terminates fatally through pulmonary edema.<sup>1</sup> The natural tendency of epilepsy is to dementia. The oftener the attacks occur, the worse the mental prospects. Death by status, or through vascular accident, or by suffocation, or other physical mishap attending a fit, is not so very rare. Tuberculosis is a frequent cause of death. Epilepsy, with fits at long intervals, particularly if first appearing after twenty years of age, is not inconsistent with mental qualities of a high order, and may not shorten life or abridge usefulness. The majority of epileptics, under proper conditions, may be advantageously and profitably employed in colonies provided for the purpose.

**Treatment.**—A systematic study of the patient is the first step toward treatment. Every possible source of local irritation must be

<sup>1</sup>Shesman, "N. Y. Med. Jour.," Jan. 11, 1906.

investigated. Occasionally, the aura may direct attention to some body-disturbance that acts as an inciting cause of the fits. Asthenopia due to errors of refraction, or faulty eye muscles, nasal disease, pharyngeal tumors, maxillary or dental faults, gastric inadequacy, intestinal parasites, rectal ulcers, hemorrhoids and fissures, constipation, diarrhea, vesical and genital troubles, must all be sought and corrected, if found. *Toxic conditions* are equally important. Auto-intoxication from the stomach, intestines, or kidneys must be stopped. Alcohol, lead, tobacco, coffee, tea, and dietetic errors may incite the fits in those predisposed. *Hygienic regulations* are of the greatest importance. These pertain (1) to the diet, which should be nourishing, easily digested, and of reasonable amount. Milk, fish, vegetables, and suitable fruit, with a small amount of starchy articles and a very little meat, is generally best for epileptics. In some instances an absolute milk diet is decidedly helpful, and may be continued for many months with advantage. (2) Baths and exercise to keep the skin, muscles, and circulation active, and outdoor life to further the same end, are valuable. Often vigorous manual labor is advisable. (3) The rooms, ventilation, clothing, occupation, amusements, and habits of the patient are worthy of full consideration. Do not overlook the tendency to constipation.

Improvement under such measures is the rule, and in rare instances it is one's good fortune to see epilepsy subside after the removal of some irritation or the correction of some toxic state.

Turning to drugs, the most importance attaches to the bromide, but they should be reserved as a last resort or as an adjunct. Cures by the bromid treatment are not to be expected. It is at best a palliative treatment and one often fraught with great disadvantage. Usually, in order to completely suppress the attacks an amount of bromid is required that maintains constant hebetude. As a practical fact, if the fits are not controlled by sixty grains of bromid a day, the question is quite sure to arise whether the epilepsy or the bromid is the greater evil. It is a frequent experience to see patients, brutalized by bromid, go months without fits, but with a loss of mental and physical activity. Sometimes the attacks then recur with seemingly additional violence, or status may develop. Among the bromids there is little choice, but the sodium salt is least likely to disturb the stomach. During the use of the bromids the intestinal tract must be kept aseptic by the use of large quantities of drinking water or some of the laxative waters, and the administration of some antifermentative, like salol or betanaphthol. No attempt should be made to displace nocturnal fits by giving large doses of bromid at bed-time. Rather should the nocturnal feature of the disease be favored, and in other cases it is to the advantage and safety of the patient to induce a nocturnal incidence of the attacks. To this end the bromid may best be administered in a single dose on rising in the morning, or by a dose after breakfast and at noon. When menstruation aggravates the epilepsy, the doses of bromid may be doubled for a few days before, during, and after the period.

Sometimes the use of antipyrin, phenacetin, trional, or other of the coal-tar derivatives, with bromids, gives better results than the bromids alone. Digitalis or belladonna in similar association will be found



valuable in those cases where there is weak circulation. Sumbul and sodium have their advocates, but so has had every other remedy ever known to man.

Luminal I have found of very distinct value, particularly in cases long treated with bromids or in which bromids are inefficient. One-quarter to one-half a grain seems to equal fifteen grains of the bromids, and its use is not attended by the cutaneous and mental disturbances of the bromid routine. Flechsig has proposed the use of opium, to be followed by bromids, in cases of long standing. He gives opium in doses gradually increasing from one to fifteen grains or more a day, if well borne, and after a few weeks abruptly stops the opium, substituting bromid, twenty to forty grains, three times daily. Flechteren combines bromid, alois vernalis, and codein with favorable results. Any one of these plans may be tried when bromid alone fails or loses its force.

Richet and Toulouse<sup>1</sup> called prominent attention to the asserted value of withdrawing common salt from the food of epileptics, thereby securing, it was thought, a better absorption of the bromids. A very general trial of this measure has led to the belief that it is one of decided value, and as a rule smaller doses of bromids are more efficacious under this régime than larger ones without it. Even alone, without the use of bromid, the withdrawal of salt secured a reduction of the number of fits in fourteen cases reported by Garbini, and the attacks were less severe. The plan is entitled to a trial in all cases, but is often very difficult to carry out in the usual run of private practice. The physical health declines rather rapidly if salt be entirely excluded, but a decided limitation of the amount of salt can usually be effected with advantage. Patients vary greatly in this regard as in all others, and personal peculiarities and requirements must be kept in view.

## CHAPTER XII.

### MIGRAINE.

MIGRAINE is an explosive, paroxysmal psychoneurosis. The attack, usually commencing with sensory and mental symptoms, is almost always attended by headache, which is frequently one-sided, and there is generally nausea and vomiting. It is sometimes called *hemiancra*, *sick headache*, or *neralgia*. Owing to the vomiting, it is often erroneously attributed to "biliousness."

**Etiology.**—*Heredity* is strongly marked. It is more commonly direct than in almost any other neurosis. Migraine may sometimes be traced through several generations, numbering dozens of cases in a single family tree. Any neuropathic family is almost sure to present cases of migraine. It seems capable of transmission by transmutation, alternating with hysteria, epilepsy, and insanity. It may be associated with the graver neuroses, or with psychoses, in a given patient. Gout and arthritis have similar close relations with it.

<sup>1</sup> Richet and Toulouse, Paris Academy of Sciences, Nov., 1893.

Thirty per cent. of cases begin between five and ten years of age, and the balance appear mainly at puberty, adolescence, and during early adult years. In rare instances it may begin after thirty. The female sex is somewhat more commonly affected than the male.

The exciting cause is often obscure. Some cases date from periods of *lowered physical health* arising from any cause. The cases beginning in early childhood very frequently follow the first systematic use of the eyes for near vision, as in school-work. *Eye-strain*, arising from accommodative or muscular asthenopia, is certainly competent to excite migrainous attacks in those predisposed. Gouty or *fibrositic* conditions, *constipation*, *indigestion*, *fatigue*, *hysteria*, *emotional disturbance*, or any *febrile* *intercurrent* may set up the attack.

**Symptoms.**—The symptoms of migraine are those of the attacks. These vary considerably in different patients, but are tolerably uniform for the given case. There are usually: (1) *Premonitory symptoms*; (2) *sensory disturbances*; (3) *headache*; (4) *nausea*; (5) *vomiting*; (6) *sleep*; and (7) *complete recovery*, occurring generally in the order given. In addition, there are usually *vascular* symptoms and occasionally *mental* and *motor phenomena*.

The *premonitory symptoms* are most common in the cases in which the early sensory symptoms are best marked. For a few hours or a day the patient feels heavy, dull, apathetic, and is usually indifferent and irritable. There may be slight headache or soundness. After a nap he may wake up with a fully developed one-sided headache, or this may be present on awakening in the morning. The *sensory symptoms* occur in over half of the cases. They usually begin quite suddenly. Bright spots before the eyes, colored rings, luminous zigzags, hemianopsia, dimness of vision, clouds, etc., are some of the subjective visual disturbances. They affect both eyes, and are sometimes lessened if the eyes are closed. They are usually most pronounced or entirely confined to the homologous half fields. Some patients only feel a vague ocular discomfort, or decided photophobia may be present. Taste and hearing are exceptionally affected in a similar manner. In some cases there are sensory symptoms in the limbs, face, throat, or tongue, but especially in the hand or foot. A tingling or numbness involves the parts and gradually advances toward the center. These sensory disturbances usually last ten to twenty minutes and then subside, the headache immediately displacing them.

*Motor symptoms*, though exceptional, are very valuable indications of the cerebral nature of this neurosis. The extremity which presents tingling may show paresis, and the following headache is usually on the opposite side. Motor aphasia may be added to right-arm tingling and left hemiparesis, and the left halves of the retina may be disturbed by visual sensations, which are referred outwardly to the right fields. Temporary wood-stuffness has also been recorded. In another group of rare cases there is transient unilateral paresis of the oculo-motor, marked by ptosis, outward squint, double vision, pupillary dilatation, and loss of accommodation, constituting the so-called *spasmodic myopia*.



Slight *mood* changes occur in some patients, such as depression, mental confusion, restlessness, loss of memory, stupor, double consciousness, or a recollection of some vivid memory. Attacks of temporary delirium, with or without subsequent ignorance of the mental state, are sometimes seen.

The *headache* is the most uniform, dominant, and distressing symptom. It varies in different cases in degree, duration, and location, but is commonly intense and ordinarily circumscribed, at least at first. Often commencing as a localized, intense pain in a small spot in the temporal, frontal, ocular, or occipital region, it gradually spreads to the rest of the same side of the head, or may become diffused all over the head. Less commonly it commences on both sides as a frontal or occipital pain. Rarely, it passes down the back of the neck and into the arm. The character of the headache is tolerably uniform in the same case, but some patients have several varieties, which reappear from time to time, and are recognized as old acquaintances. The character of the attack may also undergo great modifications during the patient's lifetime. The headache lasts from one or two hours to ten, twenty, or forty, and may subside abruptly after nausea or vomiting and retching, or gradually grow less and disappear. During the height of the headache the patients usually shun light and noise, and remain as quietly recumbent as possible. Movement, such as rising or stooping, intensifies the pain. Tenderness of the scalp or nerve-trunks is unusual.

In most cases *anorexia* appears after the headache develops or has reached its height, and there is complete *anorexia*. Digestion appears to be stopped, as unchanged food is sometimes vomited many hours after its ingestion.

The *nausea* leads to *vomiting* in a fair proportion of the cases, and emesis is attended by much retching and difficulty. It is often repeated and protracted, so that biliary matter may finally appear in the ejecta. Usually, once commenced, it is provoked by swallowing any fluid, or even by the saliva, which is commonly apparently increased in amount. Often the patient is cold, pinched, clammy, and suggests the collapse of sickness or choleraic disturbance. Frequently, as the vomiting subsides, a feeling of great relief is experienced, the headache ceases, and the patient falls into a quiet sleep of a few minutes or several hours, from which he awakes and asks for, or tolerates, a little food.

The *vascular symptoms* are interesting, and have attracted great attention. Early in the attack, before the headache has appeared, there is frequently pallor or mottling of the face. In some instances a vivid red streak in the middle of the brow or a one-sided flush invariably appears. The pallor is succeeded by flushing, in some, and there may be general profuse perspiration. Commonly, the extremities are cold during the severe pain, the pulse sharp and retarded. Usually the contracted pupils show the participation of the cervical sympathetic. This rarely is unilateral, and may produce retraction of the eyeball. The inhibited digestion may be due to a similar angiospastic condition of the gastric arterioles. As the attack declines, the surface reddens, the pulse resumes its proper rate, the pupils relax, the pallor disappears,

and in rare cases some puffiness in the scalp has been noted. Increased diuresis may follow. In the intervals the patient may feel perfectly well.

**Course.**—Migraine has a tendency to persist for many years, when once established. Commonly, in women, after the menopause, it subsides, and it disappears in men after fifty-five or sixty. The attacks occur with more or less regularity, and sometimes with remarkable periodicity. Menstruation may provoke it monthly in women. It sometimes occurs every Sunday, especially in men who change their daily routine at that time. Irregular intervals of weeks or months may intervene, when any of the inciting causes may precipitate it; but it is usually noticeable that no inciting cause or condition, acting soon after an attack, fails to induce an immediate recurrence. It is evident that the attack has cleared the atmosphere and exhausted the susceptibility. In certain rare instances the migrainous attacks have been replaced by epileptic seizures presenting the same premonitory features. A case has been seen presenting migraine, epilepsy, and transitory mania, apparently as alternating equivalents. Kraft-Ebing<sup>1</sup> reports a number of cases in which transitory mental disorder occurred as part of, or in alternation with, migraine. The premonitory hemiplegia in a case reported by Nores became permanent. In advanced years the migraine may apparently be replaced by labyrinthine vertigo. After many attacks, some intellectual impairment has been noted. Granting the neuropathic substratum in migraine, the association or succession of other neuroses and psychoses is surprising mainly by its rarity.

**Pathology.**—In the absence of knowledge regarding the morbid anatomy of migraine, we are thrown back upon theories and analogies. Attracted by the vasomotor symptoms, many attributed the migrainous attacks to disturbance of the sympathetic. This is a clear confusion of effect and cause, of symptom and disease. Taking into consideration the cortical features manifest in sensory disturbance, hemiplegia, tingling, aphasia, motor loss, crossed hemiparesis, mental features, cardiac and digestive inhibition, and the vasomotor disturbance itself, there can be little doubt that migraine is a cerebral disorder. Its resemblance to epilepsy, if not its actual relationship,<sup>2</sup> points to the same conclusion. The exact nature of the cortical instability is for the future to reveal.

**Diagnosis.**—The diagnosis of migraine depends mainly upon its paroxysmal and recurrent character and its definite clinical features. The sensory premonitions and vasomotor phenomena are very significant when present. In cases presenting migrainous attacks of the milder sort, it is difficult to be sure that the headache is not due to some ordinary cause, until its repeated recurrence under somewhat similar circumstances declares its nature. "Sick headache" is almost invariably migraine. The vesperal headaches of *syphilis*, and the quotidian or tertian headaches of malaria, occur with greater periodical regularity and with much shorter intervals than the attacks of migraine. Both lack the nausea, vasomotor symptoms, and complete recovery. From *périt*

<sup>1</sup> "Allgemein und Neurologisch," Jan., 1906.

<sup>2</sup> Wilfred Harris, "Transient Hemiplegia," "Epilepsy," 1897.



and the diagnosis may often offer considerable difficulty. The premonitory sensations may be taken for an aura, but their prolonged duration is unlike the momentary warning of epilepsy. Unconsciousness does not occur in migraine; it is the most constant feature of epilepsy. Headache, as a symptom of other disease developing in a migrainous patient, may be overlooked and the concurrent malady neglected. The headaches of Bright's disease, of cerebral tumor, of syphilis, or malaria may be wrongly attributed to the neurosis. Sensory, aphasic, and parietic features sometimes suggest Jacksonian attacks and focal brain disease. A careful study of the case and the absence of other evidence of organic lesion will usually suffice.

**Prognosis.**—Migraine is usually a stubborn and persistent malady. It has a tendency to last until involutional changes in the organism commence, when it frequently spontaneously subsides by a gradual increase of the intervals between the attacks, rather than by a lessening of their severity. If the disease is of short duration, and some removable cause can be discovered, the prognosis is fairly good. In older cases the attacks can usually be rendered less frequent and often aborted if the patient is watchful and persists in treatment.

**Treatment.**—Unless the condition or agent provocative of the attacks can be discovered and removed, there is little likelihood of fully successful management. To this end all sources of peripheral irritation and auto-intoxication must be carefully investigated. Eye-strain, improper diet, excesses, or bad habits of any sort must be corrected. In some migrainous patients any relative excess of nitrogenous food is sure to induce an attack. As a rule, for these patients an abundance of outdoor air, free cutaneous and intestinal excretion, and an unstimulating diet are indicated. A tablet of  $\frac{1}{100}$  of a grain of nitroglycerin, allowed to dissolve in the mouth, and taken at the earliest premonition, will sometimes abort an attack. Caffein has a similar effect with some patients. Others, by taking a large dose of brandy and lying down, occasionally escape. Others, again, by inducing emesis, or by washing out the stomach, interrupt the paroxysm. When the attack is under on, heat or cold to the head, warmth to the extremities, and a mild sinapism over the stomach are helpful. A dark, quiet room is usually demanded. Morphine will control the pain, but should, if possible, be avoided, to prevent setting up the opium-habit.

If the attacks are of considerable frequency,—say one or two a week,—a continuous course of brandy, as in epilepsy, may give good results. In the forms marked by parietic symptoms of onset—by ophthalmoplegia, for instance—the iodine treatment is of the greatest value, and such cases should be managed much as cases of epilepsy with incomplete attacks. Cannabis indica in association with the bromids or alone sometimes is of real value. A reliable fluid extract may be used. Beginning with 2 drops after every meal, the dose may be increased 1 drop daily until 15 or 20 drops are taken three times a day, and then reduced in the same way, and the course repeated after an interval of a week.

## CHAPTER XIII.

## NEUROSES FOLLOWING TRAUMATISM.

THE subject of neuroses following injuries is one of vast importance to the general practitioner, and has a medicolegal side of much interest. The question has been greatly belagged for many reasons. The first important work directing attention to this class of cases was the publication of Erichsen in 1871 on "Spinal Concussion," reprinted in 1875. In it he reports fifty-three cases resulting from injuries, received mainly upon railways. These cases present all manner of lesions, above lying in common the negative feature—absence of external evidence of injury. In this list are embraced cases of fracture of the dorsal vertebra, hemorrhage into the cord, division of the cord, meningitis, simple neuritis, hysteria, neurasthenia, and pretty much everything else. Unfortunately, "concussion of the spine," as set forth by Erichsen, was seized upon by lawyers as the basis for prosecuting suits for damage against corporations, and it became fixed as a diagnosis in the legal and medical mind. In 1883 Page, a railway surgeon, brought out a book written from an *ex parte* standpoint to counteract the contentions of Erichsen, and the battle raged fiercely from both sides for a number of years. Subsequently, in Germany, Oppenheim, in 1889, made a closer classification between organic injuries and those not marked by gross histological changes, and proposed the term "traumatic neurosis" for the latter. About the same time, and subsequently, Charcot taught and demonstrated that the nervous symptoms in these cases, apart from those attributable to organic lesions, were precisely the same as are presented in neurasthenia and hysteria.

The early errors of Page and Erichsen are easily understood if it is recollected that the finer anatomy of the nervous system, as well as the physiology of the cord, was but little known in their time; that the electrical reactions of nerves and muscles were not clearly understood; that the stigmata of hysteria and the symptom-group of neurasthenia were still undisciplined. Unfortunately, there was a tendency to dignify all the morbid conditions following serious injury by such terms as "spinal concussion," "railway spine," "traumatic neurosis," and "Erichsen's disease," without any attempt to distinguish their real significance or differentiate among them. The great war gave rise to the term "shell shock," under which was embraced the same category of psychoneuroses.

It must be evident that after shock with or without physical injury three classes of conditions may arise: (1) All sorts of injuries of a surgical character; (2) traumatic hysteria; and (3) traumatic neurasthenia. Various combinations of these three may result, and they are commonly found in association. Surgical conditions and neurasthenia or hysteria-neurasthenia may be present, or neurasthenia or hysteria may alone follow concussions, injuries, or frights attending accidents. It should be clearly recognized that the nervous disturbances marking neurasthenia and hysteria are likely to develop in proportion to the predisposing tendency in the individual, and also in proportion to the amount of mental shock attending the accident. In railway accidents



and war the element of fright reaches its highest development, and consequently there is a preponderance of neurosthenia and hysteria, or their combinations, in persons the victims of such accidents.

In the consideration of a case in which physical conditions and nervous symptoms have originated from injury, it is necessary to look at it first as a surgical case, and secondly to consider it as a nervous case. Cranial fracture, cerebral hemorrhage, focal epilepsy, or traumatic insanity may follow injuries to the head. Dislocations and fractures of the spine, lacerations and hemorrhages of the cord, myelitis and meningitis, muscular strains, and ligamentous ruptures may follow blows and injuries to the back, either directly or indirectly applied. Concussion, if sufficiently severe, even without apparent local physical injury, may induce hemorrhages in the meninges or in the cerebrospinal apparatus. It would be well to drop the terms *concussion of the brain* and *spinal concussion*, as there is a tendency to look upon them as vague but actual diseases. Concussion is a mode of action only, and like any other manifestation of force may vary in every degree so as to be entirely insignificant or of the utmost gravity. Accidents producing such injuries may at the same time so disturb the nervous equilibrium that neurosthenia is developed or hysteria is provoked. The neurosthenia of traumatism, or of fright associated with the possibilities of traumatism, is exactly the same as neurosthenia arising from any other source. Hysteria associated with traumatism, or conditions associated with traumatism, is exactly the same as hysteria occurring from other causes. The combinations of organic with nervous diseases of a character not yet associated with known organic changes must be deciphered on distinct lines. The surgical features have their own prognosis; the nervous disorders have their proper outlook, and they are not necessarily related.

Cases of this character may be considerably complicated by litigation and pension or indemnity claims. In exceptional instances there is dishonest and outright simulation. More frequently the anticipation of legal proceedings, the numerous special examinations, the suggestions arising from attorneys and physicians, and the very natural tendency to exaggeration serve to highly accentuate the subjective side of the clinical picture. Corporations and their legal and medical officers usually look upon all such claimants as dishonest, and by their bearing, if not by their words, antagonize and aggravate the patients who come to take an almost morbid, spiteful pleasure in cultivating their aches and bodily and mental distress. They see damages in every symptom, and the hopeful expectation of physical recovery that is so potent for good is completely destroyed. This peculiar, morose, depressed, and querulous mental attitude has even been dignified by the names *litigation psychosis* and *traumatic psychosis*. It not infrequently results that, upon the completion of litigation and the cessation of irritation and introspection, prompt improvement takes place. From a medical standpoint, it is always better that an immediate legal settlement be made.

The profession should recognize that traumatic neurosthenia and traumatic hysteria are serious and disabling conditions. Every case must be specialized, and the amount of disability and the probability of its duration must be estimated from all the facts.

## PART VIII.

### SYMPTOMATIC DISORDERS.

---

THERE are a number of symptomatic disorders very commonly met with in nervous maladies and essentially nervous in character that nearly reach the dignity of diseases. Headache, vertigo, insomnia, and neuralgia are the most important. Headache and vertigo have been specifically emphasized whenever they had a special relation to diseases of the nervous apparatus, and can be easily followed from the index. Neuralgia and sleep disorders require further study.

#### CHAPTER I.

#### NEURALGIA.

NEURALGIA (nerve pain) is always a symptom. Difficulty arises from the fact that intense neuralgic affections have often been confused with their most prominent painful symptoms. Neuritis and neuralgia have been differentiated only in recent years. Even now there are many who fail to discriminate between trifacial, brachial, and sciatic neuritides, and neuralgias of similar location. It also appears proven that an irritation at first producing neuralgia may, by its persistence, set up a neuritis or contrariwise, and it is certainly clear that neuralgia is one of the symptoms of neuritis. Gordon,<sup>1</sup> basing his conclusions upon the histological examination of nerves removed in eight cases of facial neuralgia, says:

(1) The occurrence of degeneration of the peripheral nerve is frequent if not constant in neuralgia.

(2) That this nerve degeneration is very probably a primary condition, which as a neuritis assumes an ascending course and involves secondarily the Gasserian ganglion. Although this contention is still debatable, there is great probability in favor of the above view.

<sup>1</sup>"N. Y. Med. Jour." July 21, 1906.



(3) The blood-vessels undoubtedly play a certain rôle in the causation of a degenerative state of the peripheral nerve.

(4) That it is difficult if not impossible to draw a sharp distinction between neuritis and neuralgia, as accumulated facts show an anatomical basis in the latter affection.

(5) In view of these anatomical facts, it is highly important to remove surgically a nerve affected with so-called neuralgia as early as possible after a short trial of medical treatment is given.

The distinction, therefore, is not always easy. Neuralgia may be an expression of disturbance acting (1) locally, or (2) systemically, or (3) in both ways at once. Neuralgias may be visceral or peripheral, and many visceral diseases have their corresponding peripheral or somatic neuralgias. In this relation the reader is referred to the section on Pain and the Referred Pains of Visceral Disorders as outlined by Head (p. 57 of seq.). Many neuralgias—such as angina pectoris in cardiac disease, intercostal neuralgia in gastropyloric disease, testicular neuralgia in kidney disease, pleurodynia, gastrodynia, and coccygodynia—are very clearly symptomatic and secondary. There are certain conditions favoring the appearance of neuralgia, and there are a few localized neuralgias that require individual consideration.

**Conditions Favoring Neuralgias.**—Intense neuralgic conditions are rare in early life and in old age, but when they appear after sixty, they usually have an irremediable organic basis and are correspondingly intractable. Women suffer in this way more than men. In some instances there is a marked hereditary tendency, and, as a rule, the "nervous" and neuritic, the possessors of unstable nerve-cells, are most prone to neuralgic conditions. This shows out strongly in the clinical histories of hysteria and neurasthenia. Arthritis in its broadest sense is a congener.

The inciting causes of disturbance marked by neuralgic features are very numerous. Any impairment of health, any debilitating influence, any continuous fatigue, anemia, emotion, concussion, may be symptomatized by neuralgia. Exposure to cold, peripheral local irritations, and any cause of neuritis, as well as every form of neuritis, may set up neuralgic pains. Scarcely all toxic influences may induce neuralgia. Those toxic causes which are so active in the etiology of neuritis may induce neuralgic pain. Lead, alcohol, diabetes, auto-intoxication, malaria, and acute infections may all have neuralgic symptoms. The root pain of tabes, the peripheral pains of cortical brain disease, and the nerve pains of neuromata are easily understood.

**Characters of Neuralgic Pains.**—Neuralgic pains are commonly unilateral, and confined to the distribution of a part or the whole of a single nerve, or of several nerves. The pain is inconstant and usually paroxysmal, with complete freedom in the intervals, or with a dull ache between the severe pains. They are usually described as darting, stabbing, tearing, ripping, lightning-like, as if shot, and by other words or phrases expressing their sudden, instantaneous character. They may be repeated rapidly during a few minutes, or occur singly at longer intervals. The pain is located deeply in the parts, but often

is attended by great superficial hypersensitiveness. At the same time deep pressure may give relief. During the painful attacks, motion of the muscles of the parts, a touch, even a breath of air in severe cases, may renew the stabbing pains. Heat, cold, and alcoholics may intensify or diminish the pain in various cases.

Neuralgic areas during the pains and between the paroxysms usually present nerve tenderness. This is most prominent at certain points where the nerve is superficial, overlies bone, or is inclosed by fascia or other rigid tissue. They correspond to the "maxima" of Head and constitute the "tender points" of Valleix.

In some cases the pain has a tendency to radiate into other branches of the same nerve or into related nerves. In the same way irritation of one branch of a nerve, especially of the trifacial, may induce neuralgia in another division. Neuralgic pains are often very fugacious, appearing now here, now there, especially when arising from systemic causes.

The associated muscles may act spasmodically in the pain storms. This is rather common in the trifacial form, but it is difficult to tell whether the grimace is volitional or not. In the same way a sharp leg neuralgia may be attended by a drawing up of the limb, and, if the patient is walking, he may suddenly fall.

Very often the parts subject to neuralgia show vasomotor disturbance and trophic changes. The vessels, at first constricted, usually dilate, and flushing follows. Edema, local sweating, erythema, scabiness, loss of hair, blanching of the hair, herpes, and pigmentation may attend upon neuralgia that is symptomatic of a neuritis. Localized hypertrophy, due to the continued congestive features of neuralgia, may be encountered.

**Pathology.**—The mechanism of neuralgic pains has been a fruitful theme of discussions, into which we need not enter. The following facts indicate the interposition of the spinal apparatus in neuralgic pains: (1) The pain may occupy the areas related to several spinal segments, and not closely follow the distribution of nerves. In shingles, for instance, the herpetic and painful area on the upper trunk is bounded by horizontal planes, and not by the intercostal furrows; (2) irritation of one branch of the nerve may be radiated into another, which it could only reach by way of the nucleus cells; (3) the pain may develop exclusively in another region than the one irritated; (4) division of the posterior nerve-root, as has been done, especially by Abbé and others, or separation of the afferent path anywhere between the cord and lesion, immediately stops the neuralgia; (5) irritation of the proximal stump of a divided nerve gives rise to pain referred to the periphery to which that nerve is anastomically related. This is seen in amputation neurotoma and in anesthetic disorders.

By this conception we are able to understand how systemic poisoning, as by alcohol or malaria, may so predispose the spinal ganglia that pain is occasioned by a peripheral disturbance, perhaps insignificant in itself. It also explains the ability of an intense or protracted peripheral irritation to set up localized pain, which may long persist after the irritant condition has subsided.



**Varieties of Neuralgic Pains.**—An enumeration of the common varieties of neuralgic pains is all that need be attempted. They are classified (1) as to location, and (2) as to cause.

*Varieties Depending upon Location.*—Trifacial, cervico-occipital, cervicobrachial, brachial, dorso-intercostal, intercostal, lumbal-abdominal, spinal, sacral, coccygeal, sciatic, crural, metatarsalgia, etc. Visceral forms: Pleurodynia, angina pectoris, cardiacgia, gastralgia, gastrodynia, hepatalgia, enteralgia, nephralgia, cranialgia, testicular neuralgia, etc.

*Varieties Depending on Cause or Association.*—Epileptiform neuralgia, really a neuralgic facial tic; reflex sympathetic neuralgia, one in which the pain appears at a distance from its irritant source; traumatic neuralgia, really traumatic neuritis; occupation neuralgia, a part of occupation or fatigue neuritis; herpetic neuralgia, the neuralgic pains attending zoster; hysterical neuralgia, really stigmata of the neurosis; rheumatic, gouty, diabetic, zemic, and malarial neuralgia, associated with, and often due to, the respective systemic states; syphilitic neuralgia, very rare, and due to the syphilitic cachexia. Pains in syphilis are ordinarily due to neoplastic infiltration of the nerves and other tissues; degeneration neuralgia appears in the aged, and is due to involutional changes in the organism.

**Trifacial Neuralgia.**—Of all varieties of neuralgia, that occurring in the trifacial is the most important. It often is extremely persistent and intractable. The constant exposure of the fifth pair in the face and nasopharynx to injury and infection of the periphery, the course traversed by the nerve through bony channels and over resisting structures, and its very extensive distribution territory, lay it especially liable to irritating and traumatic conditions. Its association with other cranial nerves sometimes causes it to be reflexly affected, as from the motor oculi and pneumogastric. It is affected about equally often on either side, and very rarely bilaterally. Exceptionally, all three branches are painful, but more commonly the neuralgia is confined to one or two of them.

When the *first branch* is affected, the pain is supraorbital, radiating from the supraorbital foramen over the corresponding side of the brow, or even to the vertex. The eyeball is frequently tender, or may be the seat of neuralgic pains. Tender points are usually found at the notch, on the upper lid, and over the lower margin of the nasal bone. When the *second division* is affected, the pain is located over the cheek, between the orbit and the mouth, spreading onto the wing of the nose. The tender points are at the lower border of the nasal bone, over the malar prominence, at the infraorbital foramen, on the gum above the canine tooth, and sometimes on the hard palate. In neuralgia of the *third division*, pain traverses the lower jaw and tongue and the corresponding portion of the face, extending, by the auricular branches, to the zygomatic, and even to the parietal region. The tender points are over the inferior dental foramen, in the temple, and in the parietal regions.

The pain is usually intense, lancing, shock-like, and may cause the most excruciating torture. The attacks, if at all severe, usually cause vasomotor and secretory disturbance. Lacrimation, salivation, and mucous discharge from the nose may be encountered. The brow, or

lip, or tongue, or the entire side of the face, may be swollen and edematous. The hyperalgesia is often intense, so that wiping the nose or eye, taking liquids into the mouth, and mastication are attended by great suffering, and often provoke a repetition of the neuralgic pains. Herpes possibly only occurs when histological changes in the nerve or its nucleus have taken place.

The neuralgias of the buccial, intercostal, and sciatic nerves are often intense, and present similar tender points and superficial hyperalgesia. The location of both have been described on page 57 *et seq.*

**Treatment.**—The treatment of a symptom is necessarily the treatment of the underlying disease. Local and constitutional conditions capable of determining neuralgic pains must be carefully sought, and local conditions capable of producing neuralgia at a distance must not be overlooked. In order to specialize the matter we may consider the treatment of trifacial neuralgia in detail. With proper variations the same considerations and measures apply to other neuralgias.

**Treatment of Trifacial Neuralgia.**—In the treatment of a trifacial neuralgia a careful search for local irritation is first to be made. In many cases pressure upon a given point will check or inhibit the pain. If such an inhibiting point can be found, it is a source of great relief to the patient. If the pain is intense and the hyperalgesia severe, the use of morphin or cocaine may be required to make the examination. This should commence at the vertex. The scalp and brow should be carefully palpated, the orbit investigated, the eye examined for refractive errors, local inflammation, and glaucoma. The nasal fossa, the antra, the nasopharynx, the mouth, and especially the jaws, must be thoroughly scrutinized. It is useless to sacrifice teeth unless a competent dentist finds them diseased. As a rule, a careful dental overhauling is a necessary measure in protracted cases, even where the pain is not located in the dental branches.

The general systemic state is of equal importance. The facial neuralgias of infectious, grip, malaria, and over-exhaustion commonly involve the ophthalmic division. Dental and maxillary disease is most common in the middle branch. Compression of the nerve in the dental canal often causes neuralgia of the third branch. In elderly people who have lost their teeth the resulting greater elevation of the chin stretches the dental branch of the third, and may cause a neuralgia that can be readily relieved by the use of dental plates of proper vertical projections to prevent the tug upon the affected nerve. Anæmia, cachectic states, and conditions of auto-intoxication from the kidneys, stomach, or intestines, must be corrected. Neoplasms in the cranial cavity, or facial fossa, jaws, and antra, may impinge upon the nerve and set up neuralgic pains.

In the management of these cases it is usually necessary to maintain complete rest. Many cases, otherwise rebellious, improve very rapidly under the Mitchell rest system. Food must usually be taken in a liquid form, and in very severe cases the nasal tube must be employed. Mastication ordinarily provokes the pains. Directed against the pain we have in malarial cases to employ large doses of quinia, or Warburg's tinc-



ture, or both, for several days, and follow them with arsenic, iron, and quinin in moderate doses for weeks. The purpose is to cinchonaize the patient and maintain a saturation of the blood with quinin. A preparatory calomel purge is of importance. In other cases we have to resort to sedatives, and are usually reduced finally to the use of morphia. The reliable acemith of Dupresnel, in doses of  $\frac{1}{12}$  of a grain, several times a day, is sometimes of great service, and may be increased if well borne. Slight numbness of the lips, tongue, and fingers may be expected, and this amount of action can not safely be exceeded. Cocain, by local hypodermatic administration or nodal diffusion, is not reliable, and often acts badly. Morphia should only be given by the physician or a competent nurse. It is well if the patient can be kept in ignorance of the nature of the drug, owing to the tendency which is especially strong in these neurotic cases to the requirement of the opium-habit. The use of electricity is more often disappointing than otherwise. The positive pole to the tender area, with three to ten milliamperes uninterrupted current for ten minutes, sometimes allays the pain. The current should commence from zero, slowly increase until felt as "warm," and finally decrease in the same way, without shock. The use of strychnin hypodermatically in large, repeated, and increasing doses, the patient being meanwhile confined to bed and carefully fed, as advocated by Dana, is a promising line of treatment. Commencing with  $\frac{1}{2}$  of a grain every four hours, doses of  $\frac{1}{4}$  of a grain are sometimes well tolerated. It is well to employ general massage at the same time, and, as the pain subsides, massage and vibrations to the tender area and sensitive points may be added with advantage. As a last resort, operation may be advised, but only when a careful, intelligent, and persistent use of general and special measures has failed, or there is evidence of organic changes in the nerve or nuclei.

There are three general varieties of operations upon the trifacial: (1) Those for the purpose of section, excision, stretching, dividing, or twisting out the various branches at some point below the floor of the skull; (2) those directed to caudation of the trifacial ganglion; (3) division of the sensory root of the fifth above the ganglion. Operations upon the ganglion are very difficult and dangerous. Frequently, the eye on the same side has been lost. The operation of Horsley, in which the skull is opened and the sensory root divided beneath the pons, is much less mutilating and disfiguring, but seems to be attended by danger to life. After root divisions the fibers degenerate upward,<sup>1</sup> thereby producing a permanent result. This operation is worthy of more frequent employment, in spite of its difficulty and danger. Spiller and Frazier<sup>2</sup> modify this proceeding by reaching the sensory root above the ganglion but outside the skull, the operation being carried out precisely on the lines of excision of the ganglion. Operations on the branches below the ganglion usually give temporary freedom from pain, but relapses are common in the same or in adjoining branches. (For details the student is referred to surgical works.) Fortunately, with proper systemic and local treatment these severe

<sup>1</sup> L. F. Barker, "Journ. Am. Med. Association," May 5, 1901.

<sup>2</sup> "Paola Med. Jour.," Dec. 14, 1900, and *ibid.*, vol. 25, 1902.

operations are very rarely demanded. As the slighter ones of neurctomy, etc., occasionally give permanent relief, they should be tried first. Since Schloesser in 1900 began the treatment of these neuralgias by the intraneural or perineural injection of 80 per cent. absolute alcohol, reports have come from many sources tending to show that when well executed the measure is nearly always promptly palliative and very frequently curative for a period of months or years. The method consists in reaching the involved branches, as near their exits from the skull as possible, by means of a blunt, hollow needle, and injecting into or around them one to two c.c. of the alcoholic solution. Peripheral tingling and numbness show that the proper location has been reached, and after a few days sensation returns. The injection sometimes requires several repetitions at short or lengthy intervals. An excellent résumé of the subject and technique has been published by Hecht.<sup>1</sup> This method should always be employed before resorting to cutting operations. A modification of this plan of treatment employs a long flexible needle with which an attempt is made to reach the ganglion through the foramen ovale, but its difficulties and dangers do not commend it.

Intense and intractable intercostal and brachial neuralgias justify opening the spine by laminectomy and the division of the corresponding posterior spinal nerve roots. This operation has brilliant results to its credit, but in the pains and crises of tabes has as often been disappointing as beneficial.

## CHAPTER II. DISORDERS OF SLEEP.

THE disorders and disturbances of sleep, while mainly symptomatic, in some instances reach an important development and almost attain the dignity of a disease. Sleep may be defined as a recurring, necessary state of lessened muscular, mental, and organic activity, attended by comparative unconsciousness of surroundings. No physical or mental function is absolutely aleepant. Respiration, circulation, metabolism, catabolism, muscular movements, and dreams demonstrate the persistence of functional activities. Entirely dreamless sleep probably does not occur. Sleep, therefore, is a composite which may be variously disintegrated. Sleep-walking, talking in the sleep, nightmare, night-terrors, and nocturnal enuresis, may be considered as localized or partial sleeplessness. In somnambulism the motor apparatus is awake, as it is in night-terrors. In enuresis we have a somnambulism of the lumbar cord, to adopt a term from the French. Of the physiology of sleep, though it is more necessary to life than food, we know comparatively little.

**Physical Features of Sleep.**—In sleep there is muscular relaxation. The lids are lowered over the upturned eyeballs; the expression is one of repose. Respiration is slower and less deep. Mosso states that the

<sup>1</sup> Jour. Amer. Med. Assoc., Nov. 9, 1907.



amount of air inspired by a normal man during sleep is one-seventh of that used during similar periods of quiet wakefulness. Breathing is distinctly thoracic in character, the diaphragm acting but slightly; inspiration is more prolonged and the respiratory pause is absent. There is a decrease in carbonic acid elimination and an increase in the absorption of oxygen. The circulation presents important modifications. The pulse is less rapid. The superficial of the body has an increased vascularity and is often reddened; there is lowered arterial pressure and a smaller central circulation.<sup>1</sup> The brain is comparatively anemic. The arterial changes take place in a precise way, increasing with some rapidity during the first hour, maintaining a maximum for an hour or two, and then gradually decreasing to the waking moment. Such curves have been shown by the plethysmograph and correspond fairly well to those indicating the depth of sleep as demonstrated by Kold-schütter and others. In accordance with universal experience, the first few hours of sleep are, therefore, most profound, refreshing, and valuable. The thorax and limbs actually increase in size during sleep, owing to the circulatory conditions; and we readily comprehend the increased activity of the skin, the tendency to night-sweats, and the ease with which one is chilled during sleep. Lombard, Rosenbach, and others have observed that the muscle reflexes are exaggerated just before

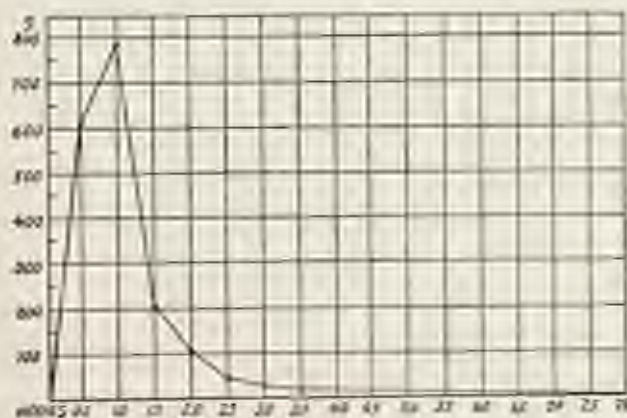


FIG. 208.—Curve illustrating strength of voluntary stimulus (falling ball) necessary to arouse a sleeping person. The curve was marked before, and the time was noted at habitually asleep. The curve shows that the height from which the ball must be dropped just before awakening at the end of the first hour (Koldschütter).

and during the early moments of sleep, but gradually subside, and the knee-jerk may even disappear. Slight disturbances tending to awaken the individual are attended by a corresponding increase in the reflex activity and the central circulation. We are all familiar with sudden startings when dropping off to sleep—a manifestation of the increased reflex excitability, constituting one of the phenomena of sleep. The pupils are contracted in proportion to the profundity of the sleep.

**Requirements for Sleep.**—Individuals vary greatly in the amount of sleep required, and at different ages there are different needs. While

<sup>1</sup> Bowdell, "Jour. of Experimental Med.," 1897.

some few adults find four or five hours sufficient, the great majority demand eight or ten. There are also temperamental differences and many variations, the result of habit and circumstances. Crichton Brown says that at four years twelve hours' sleep are needed; at fourteen years, ten hours; at seventeen, nine and one-half hours; then seven or eight hours during adult life. In old age continuous sleep is rare, and the requirement is less; but frequent naps and dozing, by day as well as at night, maintain a fair average. Infants may sleep most of the twenty-four hours. More sleep is required in cold than in temperate or warm countries.

**Conditions Favoring Sleep.**—A cool, dark, quiet, well-ventilated room, a comfortable bed, and adequate, not excessive, covering, are conducive to sleep. A preparatory period of sleepiness is natural, and, in cases of insomnia, must be cultivated. Active emotions, mental worries, intense thought, cold extremities, or a chilled skin defeat the rearrangement of the circulation, on which so much depends. No physical function is so readily disturbed as sleep. If a person is awakened at an unusual hour several nights in succession, he tends to establish a habit of awakening at that hour. Habit is all-powerful, both for good and evil, in this matter. Regular hours for retiring and waking are most important. The use of hypnotics to put patients to sleep regularly for a few nights do good mainly by reestablishing the natural initiative.

Disturbances of sleep are (1) those presenting a deficiency, and (2) those marked by a torrid increase of sleep.

#### WAKEFUL DISORDERS OF SLEEP.

**Insomnia.**—Insomnia is a symptomatic condition, marked by more or less inability to sleep the individual's usual required length of time.

**Etiology.**—The causes of sleeplessness are numerous. Practically, every deviation from health is marked by disturbance of sleep; but in many instances, once a bad sleep habit is established, it tends to persist, and may be the chief complaint of the patient. Many persons are hereditarily poor sleepers. In such instances every trivial sound or unusual circumstance—a light, an odor, a jar, or even the discontinuance of a customary noise or light—may arouse them. Hunger, over-feeding, indigestion, constipation, intestinal worms, lithemia, uremia, various drugs, tea, coffee, tobacco, alcoholism, fevers of all sorts, malaria, syphilis, lead-poisoning, cerebral hyperemia or anemia, as from cardiac disease or diathetic states, psychic disturbances, discomfort, pain, neurasthenia, grief, worry, old age, mental preoccupation, and intense study are among the principal inviting causes of sleeplessness.

**Symptoms.**—Some patients readily fall asleep, but shortly awake, and are then sleepless the balance of the night, or merely secure fitful periods of sleep. Others spend several hours getting to sleep, and may then rest fairly well. Still others complain of broken sleep, the night being passed in intervals of sleep and wakefulness, which may be quite uniform in a given instance. As a rule, patients troubled with insomnia



are disposed to exaggerate the amount of sleeplessness, and it is a common experience to find such patients sleeping soundly at times, or even for much of the night, if they are put under watch. Much loss of sleep manifests itself in a haggard, weary air, and in lessened muscular and mental force. Appetite, digestion, energy, courage, and good nature are diminished. The patient loses weight, and, in cases of absolute sleeplessness, the loss may equal that due to deprivation of food. The eyes lose their clearness and look dull, and the sclerotic may be congested. The tongue is coated, constipation may be present, and the entire organism is deranged.

**Treatment.**—The treatment of the symptom *insomnia* implies the management of the basic condition of which it is a manifestation. All hereditary, digestive, toxic, circulatory, nervous, and reflex causes must be systematically investigated. The examination of a patient complaining of insomnia omits nothing. Very frequently, modes of living, and especially bad sleep-habits, must be corrected before any improvement is secured. The physical state often requires to be thoroughly well reestablished by baths, proper diet, exercises, and good hygiene. Attention at once falls upon the conditions favoring sleep, as previously outlined. The patient must be instructed in the importance of these minutiae, and not expect to secure complete relief by a few doses of medicine. As adjuncts, a warm bath, taken quietly at bedtime, not followed by stimulating frictions, is conducive to sleep. A cold pack or an alcohol rub acts well in some cases. The stomach should not be empty. A glass of hot milk, or milk and water, or malted milk, or hot lemonade, or even of hot water, acts beneficially in decongesting the head in cases of *neuritis*. With the hot bath it favors the dilatation of the cutaneous vessels, and establishes the circulatory conditions found in natural sleep. In the same way a pint of beer not only starts the abdominal organs, but flushes the skin. Large doses of whisky are never advisable, although sometimes efficient for a night or two. Anemic cases require cardiac stimulants and blood-makers.

Any drug that sufficiently masters the organism to produce sleep is a dangerous remedy, and should be used with circumspection, and only as a last resort. Of all the hypnotics, chloral alone is uniformly reliable. In cases attended with much nervousness it is decidedly assisted by the addition of sodium bromid in equal amount. The administration of hypnotics should be done with a definite purpose. It is important to exhibit whichever one is selected at such time that its systemic effect may be operative when the sleeplessness is due. In cases experiencing difficulty in getting to sleep chloral may be given thirty minutes before retiring. Cases awakening at one or two o'clock may be given trional, in dry powder, at bedtime, as its action is liable to be delayed for a few hours. A sufficient dose should be used to have a decided effect, repeated several nights, if its action meets the requirements, and then discontinued. In some instances three-grain doses every two hours after mid-day act much better than a large dose at bedtime. Veronal is so tardy in its action, and so depressing during the following day, that it can rarely be used with much satisfaction. If the underlying cause has been corrected,

the proper routine will thereby be reestablished in a few nights. Finally, some cases only yield after a complete change of scene. An ocean or lake voyage is especially valuable, as it is devoid of exhausting excitement and sight-seeing.

**Somnambulism.**—In sleep-walking the individual acts his part of a dream. The motor apparatus is awake and responsive to the mind. It is sleep with motor automatic activity, and presents a peculiar increase of the subjective powers of the affected person. There is often great keenness of touch and analgesia combined. The special senses may be active or not, but the patient only takes cognizance of those things which pertain to the dream-story. Usually, the pupils do not respond to light, and the face has a blank, apathetic appearance. The eyes may be open or closed. The sleep-walker has no subsequent waking recollection of his somnambulistic acts; but these may be revived or repeated in a subsequent attack. Talking in the sleep is a minor degree of somnambulism, and the state of double-consciousness may be considered as its largest development.

Somnambulism may ordinarily be considered as a neurotic stigma, and is commonly encountered in those of a neuropathic heredity. Puberty is the ordinary age for its appearance, and both sexes are then about equally affected. Later in life there are more female than male cases. The individual attack is often traceable to some mental precedent condition. Even the suggestion of sleep-walking, in the discussion of the subject, has led to its appearance. In other cases the patient carries out in sleep the line of action on which he had been intent before retiring. In most cases presenting repeated attacks there is a similarity of action in all of them, or one attack may continue the action of its predecessor.

The treatment of the condition should be broad enough to cover the neuropathic make-up of the patient, and is most successful in proportion as it is directed to the mental element in the disturbance. If the patient takes with him to bed a firm intention not to walk, it is often sufficient to inhibit the attack. In nervous subjects of impaired self-control a vigorous suggestion, that will be operative during sleep, must be implanted. This, in children, may be accomplished by a system of rewards or deprivations, or a cold spinal douche at bedtime, associated with the suggestion that it is to prevent walking during the night, or by an emphatic admonition from a respected source. The individual attack may be brought to an end by a dash of cold water, or a sharp puff of breath in the face, or by firm pressure over the suprasternal foramina. So mild a shock may be curative, but in highly nervous children is to be used with caution. In adult cases, with frequently repeated attacks, any plan of treatment is likely to fail, and in some instances nightly precautions are necessary to guard against accidents.

**Dreams.**—Dreaming is perfectly physiological. As already stated, it is probable that dreamless sleep does not occur. Only those dreams, however, are remembered that are particularly vivid or occur during light sleep, the ordinary condition after the third hour. In numerous experiments it has been found that sleepers, suddenly and unexpectedly aroused, can always give an account of the interrupted dream. Dreams,



however, have a considerable medical importance. In lowered nervous or physical states the sleep is not profound, and the patient complains of dreaming constantly. The nature of the dreams has a general relation to the physical and mental status. Only the well have concerned, happy dreams. Neurasthenics, hysterics, and melancholics are tormented by troubled dreams of an unhappy, depressing character. Neurasthenics and hysterics are prone to have some particular, formulated dreams that occur several times in a night or on different nights. The influence of a terrifying dream in hysteria may equal a severe mental shock in the waking moments, and may be the basis of a hysterical, fixed idea, leading to paralysis, anesthesia, contracture, or assertion of attempts upon elasticity (Janet).

In some nervous conditions the dream state is projected into the waking state for a few moments, or for a longer time. In hysteria the features of the dream may then persist for an hour or more, constituting a delirious accident. It is a sort of somnambulism, but with greater mental and physical activity. Or the dream may evoke a convulsive crisis. Of the same nature, perhaps, is *sleep-crashiness*, or *somnolence*, in which a person, on being suddenly awakened from deep sleep, is apparently manic, and may do acts of violence. A great interest in dreams has followed Freud's studies of the dream state. He insists that during sleep, judgment being reduced, the natural tendencies as well as the morbid ones are untempered, and subjective states prevail. By a systematic study of the dream content he thus frequently discovers the fixed or dominant idea controlling hysterical and psychasthenic manifestations.

In *pavor nocturnus*, commonly called night-terrors, and occurring only in children, the little patient awakes in vague, wild alarm one or two hours after going to sleep. The child screams in terror, clings to its mother, but apparently fails to recognize any one, and can not be quieted or reassured. After a few minutes or more the excitement spontaneously subsides and the child returns to sleep without recollection of the attack in the morning. During the frenzied terror the patient may run from the room, or climb upon the furniture, in a wild effort to escape something. Often the cries imply a fear of being caught by some one or by wild animals.

True *pavor nocturnus* is of somewhat serious import, as it indicates an unstable nervous constitution, and is a frequent item in the early history of epileptics. Some writers consider it a true neurosis, and, at least, it is a stigma of degeneracy. It is often apparently induced by respiratory difficulty dependent upon facial adenoids, bronchitis, laryngitis, weak heart, or general asthenia. In mild form it may only suggest nightmares or *visions*, which is a vivid dream, usually traceable to some physical condition or previous terrifying experience. Indigestion, bad ventilation, or mental shocks, fright, worry, etc., give rise to nightmares attended by a feeling of a great weight on the chest, suffocation or falling. Ordinarily, at such moments the patient suspends respiration, or makes distressed inspiratory noises and awakens with a start.

**Nocturnal enuresis** is a common disorder in weakly and neurotic children. Neurasthenics, hysterics, epileptics, and the entire range of neuropaths give abundant testimony on the subject of bed-wetting in

childhood. In certain instances it undoubtedly is an automatic action, a partial *somnambulism*. The patient dreams he is passing water, and voids his urine accordingly. The same mechanism may operate in the more frequent form. Parents report that when this occurs under observation the bladder acts forcibly, and the stream is propelled with vigor. It is not a mere sphincteric relaxation, as is sometimes alleged. Such children, if taken up at night and made to urinate, may repeat the act almost immediately upon being put back to bed, perhaps through suggestion. It is probable that slight distention of the bladder, or the presence of a drop or two of urine in the vesical end of the urethra, gives rise to sensations which set up a chain of ideas eventually leading to the urinary act. It is definitely proved that various external stimuli—as sounds, voices, lights, etc.—may be partially apprehended by the sleeper and woven into his dreams, materially modifying their trend. Internal sensations are no less liable to act in the same manner, and are generally accredited of doing so in nightmare.

The management of nocturnal enuresis demands attention to several factors: First, the general condition of health; second, the reduction of nervous excitability; third, the prevention of a large vesical accumulation; fourth,—and perhaps the most important of all, as in *somnambulism*,—the establishment of a definite idea of self-control. It is well to withhold liquids toward the evening, and to have the child thoroughly evacuate the bladder at bedtime. Elevation of the foot of the bed by several inches, to gravitate the urine from the neck of the bladder, has been strongly recommended by Mendelssohn. *Belladonna* reduces vesical irritability, and the urine should be rendered unarritating. Local disturbance in bladder and urethra must be removed and intestinal worms expelled. Finally, a strong mental impression is of the greatest value. For instance, Prendergast<sup>1</sup> cured seventy-five out of eighty cases in a boys' orphanage within a short period of time by giving them a cold spinal douche, followed by a quick rubbing-down just before getting into bed. Nothing else whatever was done, and the five remaining cases were much improved. In some cases the first douche had the necessary effect, and the mind remained on guard.

#### SOMNOLENT DISORDERS OF SLEEP.

The second group of sleep disorders comprises those marked by somnolence, or by an irresistible tendency to sleep.

**Narcolepsy** is a condition in which the patient repeatedly goes to sleep during the day. The tendency is irresistible, and the morbid sleep may last a few minutes or several hours. It furnishes one of the accidents of hysteria, and may be dependent upon drugs, brain disease, gout, obesity, uremia, diabetes, and syphilis. Some nervous families present a number of instances of narcolepsy, associated or alternating with *grotesque* neuroses, mainly hysteria and epilepsy. It may be an epileptic equivalent, and many cases also present epilepsy. It should always suggest the possibility of epilepsy or hysteria if defective nutri-

<sup>1</sup> *New York Med. Jour.*, July 11, 1896.



tion and the systemic states mentioned can be excluded. In some instances ocular fatigue, due to refractive errors, seems to be the starting-point for the attacks, which are overcome by appropriate glasses. In some instances the sleep in such attacks is disturbed, and the patient may have vivid dreams and talk in a flighty manner. In other cases, as in epilepsy, the patient is comatose and can not be aroused. In the majority of cases the sleep is deep and no dream recollections are obtainable.

Treatment depends upon deciphering and removing the cause, and then breaking up the habit. For this purpose caffeine and nitroglycerin or nitrate of amyl, to control the cerebral circulation, are available.

**Sleeping Sickness.**—On certain African coasts the natives are affected by a parasitic disease. Somnolence appears at intervals and gradually increases, until the patient sleeps steadily, at first arousing sufficiently to take food, and finally not awaking under any stimulus. In two cases carefully observed by Mott,<sup>1</sup> a chronic diffuse leptomeningitis and meningoencephalomyelitis were found, but no infectious organism. Later observations sufficiently confirmed show this disease to depend upon the invasion of trypanosomes inoculated by the "tsetse" fly. They cause a diffuse chronic lymphadenitis leading to a chronic interstitial inflammation of the lymphatic structures of the brain and cord. Death usually results from inanition in four or five months, but patients have lived for two or three years. Treatment by stoxyl, an arsenical preparation, appears to be specific.

**Trance, catalepsy, and ecstasy** are hysterical accidents—frictional hysterical convulsions extended into status. They are described, with their treatment, in the discussion of Hysteria.

#### SLEEP PALSES.

Persons with disordered systemic conditions—such as in menia, gout, diabetes, women at the menopause, excessive users of tobacco, etc.—may awake with benumbed extremities. The hands and feet are most affected, giving origin to the term *nyctoparesis*. The sensations are described as pricking and numbness, as being "asleep," and some cramping and decided pain may occur. A loss of power is noted if the condition is marked. The disturbance lasts a few minutes or an hour, and is usually relieved by friction and exercising the members. Less frequently one limb only is affected, or both limbs on one side. This affection may occur nightly for months and years, and appear during the day also if the patient fall asleep. Its treatment is that of the underlying state.

In sleep, especially the profound sleep of drunkenness or the coma of anesthesia, pressure on a nerve may set up a neuritic palsy. This is particularly common in the musculospiral, but in operations on the perineum and pelvic contents the lithotomy position is capable of injuriously stretching the sciatic. The brachial plexus may be injured by elevating the arm too forcibly in operation on the breast, or by the anæsthetic. These are hardly to be called sleep palsies, but are definite accidents occurring during sleep.

<sup>1</sup> *Reg. Med. Jour.*, Dec. 16, 1899.

Anæmic neurasthenic patients sometimes have difficulty in opening the eyes in the morning, but if the lids are once raised by the finger, they tend to remain open. The symptom is called *sleep ptosis*, is usually transient, and commonly passes away with the myasthenia of which it is part. This, or a similar condition, occurs as a part of myasthenia gravis or pseudobulbar paralysis, but in such cases is caused or increased by fatigue and tends to subside during sleep.

### HYPNOTISM.

Hypnotism is an artificial, morbid, sleep-like condition produced in susceptible persons by various methods. In it the complex of sleep is more or less disaggregated. Attention within a limited field and automatic motor activity may be retained, but mental initiative, volition, and judgment are much impaired. The acts and circumstances of well-marked hypnotic conditions can not be recalled by the subject unless the hypnosis is revived. It is, therefore, a state of subconsciousness or double consciousness. Different experimenters find varying proportions of susceptible individuals. The sexes are about equally hypnotizable. Those of mediocre self-consciousness, those accustomed to unquestioningly obey,—hence, children and some hysterics,—are the most ready subjects. A great deal depends upon the preparatory conditions and the belief or skepticism of the patient. In those places where daily sittings of hypnotism are held and the subject is introduced into an atmosphere of faith and observes the process in others, success in the method is naturally greatest. Experience on the part of the hypnotizer is also an important factor. The whole matter consists of building up an expectant affection. Low-grade idiots, most of the insane, and many hysterics, incapable of exercising continuous attention, are not hypnotizable by ordinary methods. The state is purely subjective to the one operated upon. After a person has once been hypnotized, he subsequently more readily enters hypnosis, and finally the condition is produced almost at a hint. In extreme cases it may be automatically evoked by anything which suggests the method employed in the given case, and the subject becomes the puppet of a trivial sound or flash of light. During hypnosis there is a high degree of suggestibility, so that the subject apparently unquestioningly accepts the dictum of the operator. In somnambulism the special senses and general sensibility may be extremely acute or entirely blunted, responding only along the various lines of the hypnotic field of activity. Autohypnosis is also possible, and is a common trick of East Indian fakirs and dime museum tricksters in this country.

**Methods of Hypnotizing.**—After the subject is prepared by example or explanation and his cooperation secured, the most common plan, that of Braid, is to cause ocular fatigue by having the patient fix the eyes in unwinking gaze upon a small, bright object held about six inches distant and above the ordinary line of vision. This shortly produces a feeling of fatigue in the ocular muscles, the eyes water, the lids tremble, and the operator, by suggesting sleep, adds to the mental



impression of drowsiness. Passes of the hand over the head or down the limbs may be added. Their soothing effect is well known, and soon the eyes close, or the lids are pressed down by the operator, who enjoins the patient to "sleep, sleep soundly, deeper yet, deep sleep," etc. Four or five minutes may be required for this performance, the patient being placed in a reclining or comfortable attitude for sleep. At this point the subject is firmly and authoritatively told that his eyes are closed, that he is asleep, that he can not open his eyes. The operator may start the subject's hands in rotation and command him to continue the movement, asserting that he can not stop until ordered, etc. Other methods contain the same elements of suggestion and are aided by the operator's voice in a similar manner. Thus, continuous gentle pressure on the eyeballs, passes, and stroking, monotonous sounds, fixing the attention, or gazing fixedly at small revolving mirrors (Lays), the patient meanwhile sitting in a darkened room, are among the methods frequently used. A loud sound or a vivid flash of light, after due preparation, may throw the subject at once into deep hypnosis (Charcot). Bernheim fixes the patient's gaze with his own and suggests sleep: "Sleep is approaching, your limbs are feeling warm and gently tingling, your eyelids are heavy, you are going to sleep; you are sleeping, sleep soundly," etc. The subject is aroused by command, by a puff of air in the face, or by stroking the head. If left to himself, he spontaneously emerges from the hypnosis after a variable time, a few minutes or an hour or two.

The hypnotic state varies in intensity from slight torpor to somnambulistic automatism. Only a small percentage of subjects can be put into the deepest phase; and it is rarely required, as, for purposes of therapeutic suggestion, the lighter phases are quite sufficient. Charcot divided the hypnotic state into three phases—the *létargic*, the *cataleptic*, and that of *somnambulism*. In the first the subject is as if asleep, without changes of respiration, pulse, or temperature. There is usually some analgesia, but the operator's voice is heard and the subject replies to questions. In the cataleptic stage the limbs retain the position given them, or, if set in motion, continue the movement indefinitely, analgesia is complete, and commands are obeyed within the cataleptic range. In somnambulism the subject is automatically responsive in every way to the operator, whom alone he sees, hears, or obeys, unless otherwise instructed. Suggestions are implicitly accepted, regardless of their non-sensical or irrational character, and commands that do not contravene deep-seated moral convictions are unquestioningly executed. Subsequently, the subject has no recollection of his automatic acts or of the conditions under which they were performed, but, rehypnotized, can readily reproduce them.

**Uses of Hypnotism.**—In spite of the extravagant claims for the therapeutic value of hypnotism, it is as yet of very little service to the physician. Now and again a minor operation may be done under its influence, saving the slight risk of surgical anesthesia, or by its aid a fixed idea may sometimes be removed and a delusion dispelled. Under ordinary circumstances the number of susceptibles is so small that its general use is impossible. In hysteria, as elsewhere, it is a two-edged

weapon, and the patient may emerge from hypnosis instituted for a minor difficulty and go into severe hysterical convulsions. One delusion may be removed, but another and more serious one of mind-reading or undue influence may be implanted. For obvious reasons, women should never be hypnotized without reliable witnesses, and the public use of hypnotism can only appeal to the mischief. In this connection its power for harm is proven. There is no longer any doubt that its frequent repetition is harmful to the individual. It tends to destroy self-reliance and to make patients imaginative, weak-minded, and neurasthenic. It also has a tendency to bring discredit upon its employer, and in most instances would better be substituted by measures of equal efficiency and less disadvantage. Suggestion, however, is a mighty aid to the physician, and, without producing hypnosis, positive and intelligent assertion can accomplish all that is likely to be done by hypnotism short of the somnambulistic stage. A fair realization of the part suggestion plays in therapeutics is one of the recent achievements of the most progressive medical minds.



# MENTAL DISEASES.

BY

FREDERICK PETERSON, M. D.,





# MENTAL DISEASES.

## CHAPTER I.

### INSANITY.

**Synonyms.**—*Psychosis, Psychopathy.* *German:* Irreiss, Irresein, Verrücktheit, Wahnsinn. *French:* Aliénation mentale, Folie.

It is the object of the author to bring together in the following chapters such matter in relation to the definition, classification, etiology, pathology, symptomatology, and treatment of insanity as will be of actual practical value to the medical student and general practitioner.

The seeker after special information and deeper knowledge of the complex subject of morbid psychology must be referred to the many profound works which deal with this exclusively. These chapters are based upon my clinical lectures given at the Vanderbilt Clinic during the past twenty years, to the students of the College of Physicians and Surgeons of Columbia University. They, therefore, embody only the facts which I believe to be the most serviceable and useful to those who are often practically concerned with the early diagnosis and prognosis of insanity, and who must be the first arbiters as to the course of cure and treatment to be pursued.

**Definition.**—The difficulty of making a rigid definition of insanity is recognized by all who have attempted it. So various are the manifestations of mental aberration, so many the faculties involved, so different the degrees of deviation from the normal, it is no wonder that the expert hesitates and often fails in the effort. The definition, too, must include idiosyncrasy, and must exclude certain states of transitory mental disorder, such as the delirium of fevers and of intoxications.

The noted English jurist, Lord Justice Blackburn, once said, while giving evidence before a committee of the House of Commons: "I have read every definition which I could meet with, and never was satisfied with one of them, and have endeavored in vain to make one satisfactory to myself. I verily believe that it is not in human power to do it."

Fortunately, we are not often called upon to give a definition of insanity, and usually we may reply that insanity is a symptom of so

many obscure pathological states, and appears in such diverse forms that a narrow definition is not possible. However, the practitioner may find himself in the witness-box some day, and it is not uncommon for one of the legal examiners to ask of the witness in a mental case a definition of insanity. If the witness be wise, he will answer as indicated, or he may qualify such answer by offering to quote some one of the definitions given by alienists, such as follow :

A disease of the brain (dilatative or sympathetic) affecting the integrity of the mind, whether marked by intellectual or emotional disorder.—(Hack Tuke.)

A special disease; a form of alienation characterized by the accidental, unconscious, and more or less permanent disturbance of the reason.—(Régis.)

Morbid derangement, generally chronic, of the supreme cerebral centers,—the gray matter of the cerebral convolutions or the *isoleucocortex cerebri*,—giving rise to perverted feeling, defective or erroneous ideation, and discordant conduct, conjointly or separately, and more or less incapacitating the individual for his due social relations.—(Maudsley.)

Insanity is either the inability of the individual to correctly register and reproduce impressions (and conceptions based on these) in sufficient number and intensity to serve as guides to actions in harmony with the individual's age, circumstances, and surroundings, and to limit himself to the registration of subjective realities of impression transmitted by the peripheral organs of sensation, or the failure to properly coordinate such impressions and to thereon frame logical conclusions and actions, these abilities and failures being in every instance considered as excluding the ordinary influences of sleep, trance, somnambulism; the common manifestations of the general neuroses, such as epilepsy, hysteria, and chorea; of febrile delirium, coma, acute intoxications, intense mental preoccupation; and the ordinary immediate effects of nervous shock and injury.—(Spitzka.)

With these few examples before us of the diversity of definition attained by careful students of psychiatry, we may well content ourselves and acknowledge that a satisfactory definition in brief form is scarcely to be devised. The writer has often qualified this by offering the following, which has at least the merit of brevity, if not of perfect adequacy :

*Insanity is a manifestation in language or conduct of disease or defect of the brain.*

The law assumes to offer certain definitions of insanity, from which, however, those of medicine would tend to differ, in connection with the three chief points where law and psychiatric medicine meet :

1. A criminal is insane if he does an act whose nature and quality he does not know, or if, knowing the nature and quality of his act, he does not know whether it is right or wrong.

2. A testator is insane if his mind, memory, or understanding is unsound.

3. In a lunacy inquisition the subject of the inquiry is insane if he



is incapable of managing himself and his affairs. Such are the divergent tests of insanity in law.

**Classification.**—What has been said of the difficulty of defining insanity is equally applicable to classification. Not all of the writers of works on psychiatry have deemed it expedient to offer a definition of insanity, but there is scarcely one who has not presented us with an original classification, or one modeled upon, or modified from, that of his favorite authority. It will be impossible as well as useless to attempt to enumerate in these pages one-half of the many classifications which have been made, held for a time, and finally abandoned with the advance of science and the accumulation of new facts in the domains of pathology and psychology. It suffices to say that there are at least forty such classifications which have been made upon etiological, psychological, symptomatological, or pathological grounds. I shall present here, simply as examples for reference, several of the latest and best classifications of the Anglo-American, German, and French schools.

The Statistical Committee of the Medico-psychological Association of Great Britain adopted the following classification for use by the medical superintendents of asylums:

1. Congenital or infantile mental deficiency—
  - a. With epilepsy.
  - b. Without epilepsy.
2. Epilepsy (acquired).
3. General paralysis of the insane.
4. Mania—recent, chronic, recurrent, *à pots*, puerperal, senile.
5. Melancholia—recent, chronic, recurrent, puerperal, senile.
6. Dementia—primary, secondary, senile, organic, —i. e., from tumors, coarse brain disease.
7. Delusional insanity.
8. Moral insanity.

Maudsley's grouping is as follows:

# 1. AFFECTIVE OR PSYCHIC INSANITY.

1. Maniacal perversion of the affective life (mania without delusions).
2. Melancholic depression without delusion (simple melancholia).
3. Moral alienation proper approaching this, but not reaching the degree of positive insanity in the insane temperament.

# II. IRRATIONAL INSANITY.

1. General
  - a. Mania
 

1. Melancholia	{ acute.
	{ chronic.
2. Partial
  - a. Monomania
  - b. Melancholia
3. Dementia
 

{ primary.
{ secondary.
4. General paralysis.
5. Imbecility.

## Classification of the Congress of Paris (1889):

1. Mania (*acute delirium*).
2. Melancholia.
3. Periodical insanity (*circular insanity, etc.*).
4. Progressive systematized insanity.
5. Venereal dementia.
6. Organic dementia.
7. Paralytic insanity.
8. Neurotic insanity (*hypochondria, hysteria, epilepsy, etc.*).
9. Toxic insanity.
10. Moral and impulsive insanity.
11. Idiot.

The following is the classification of Régis:

## I. FUNCTIONAL INSANITY.

Generalized or symptomatic	1. Mania.	Subacute mania ( <i>maniacal excitation</i> ).
		Acute mania ( <i>typical mania</i> ).
	2. Melancholia.	Hypomanic mania ( <i>acute delirium</i> ).
		Chronic mania.
	3. Insanity of double form	Remittent or intermittent mania.
		Continuous.
Partial or essential insanity.	Systematized progressive insanity	Subacute melancholia ( <i>melancholic depression</i> ).
		Acute melancholia ( <i>typical melancholia</i> ).
		Hyperacute melancholia ( <i>with stupor</i> ).
		Chronic melancholia.
		Remittent or intermittent melancholia.
		Intermittent.
		First stage ( <i>hypochondriacal insanity</i> ).
		Second stage ( <i>sensory, religious, erotic, political, etc.</i> ).
		Third stage ( <i>madness/insanity</i> ).

## II. CONSTITUTIONAL INSANITY.

Degeneracy of evolution. (Vices of or- ganization.)	Disharmony (defect of equilibrium, irregularity, eccentricity)	Neurosthenia (flood ideas, impulsions, alcoholism).
		Phrenasthenia (delirious, reasoning, instinctive).
Degeneracy of in- volution. (Disorganization.)	Monstrosities (imbodily, sensory, cerebral, myeloma).	Dementia (simple dementia).

Krafft-Ebing has drawn up this scheme:

## A. MENTAL DISORDERS OF THE DEVELOPED BRAIN.

## I. PSYCHONEUROSES.

1. Primary variable conditions.	Melancholia	simplex.
		attonita.
	Mania	Maniacal excitation.
		Acute mania.
		Stupor, or variable dementia.
		Wahnwitz (verania).



2. Secondary incurable states.  $\left\{ \begin{array}{l} \text{Secondary monomania (Verrücktheit).} \\ \text{Terminal dementia} \left\{ \begin{array}{l} \text{agitated.} \\ \text{apathetic.} \end{array} \right. \end{array} \right.$

## II. PSYCHO DYSGENERIC STATES.

1. Reasoning insanity.
2. Moral insanity.
3. Primary monomania (primitive Verrücktheit—passionary, erotic, religious, ambitions).
4. With imperative conceptions.
5. Insanity from constitutional nervous  $\left\{ \begin{array}{l} \text{epileptic.} \\ \text{hysterical.} \\ \text{hypochondriacal.} \end{array} \right.$
6. Periodical.

## III. CEREBRAL DISEASES WITH MARKED MENTAL SYMPTOMS.

1. Paralytic dementia.
2. Cerebral syphilis.
3. Chronic alcoholism.
4. Scroful dementia.
5. Acute delirium.

### B. ARRESTED CEREBRAL DEVELOPMENT.

1. Idioty.
2. Cretinism.

Ziehen has given the following classification:

## I. PSYCHOSES WITHOUT INTELLECTUAL DEFECT.

### A. Simple psychoses.

1. Affective psychoses.  $\left\{ \begin{array}{l} \text{Mania.} \\ \text{Melancholia.} \\ \text{Neurothemia.} \end{array} \right.$
2. Intellectual psychoses.  $\left\{ \begin{array}{l} \text{Stupidity.} \\ \text{Pecunia} \left\{ \begin{array}{l} \text{simple.} \\ \text{hallucinatory.} \\ \text{ideational (Ideenfluchtige).} \\ \text{stuporous.} \\ \text{incoherent.} \end{array} \right. \\ \text{Imperative conceptions.} \end{array} \right.$

### B. Mixed psychoses.

## II. PSYCHOSES WITH INTELLECTUAL DEFECT.

- a. Congenital weakness (idioty, imbecility, feeble-mindedness).
- b. Acquired weakness, or dementia.
  1. Paralytic dementia.
  2. Scroful dementia.
  3. Secondary dementia (after functional psychoses).
  4. Secondary dementia (after cerebral lesion, syphilis, etc.).
  5. Epileptic dementia.
  6. Alcoholic dementia.

Kraepelin, who has done so much of recent years to try to bring the chaos of classification of insanity into some sort of order, and who by his example has so greatly stimulated the clinical study of psychiatry, has adopted the following classification:

#### I. INFECTIOUS PSYCHOSES.

- a. Fever deliria.
- b. Deliria from infections.
- c. States of exhaustion from infections.

#### II. PSYCHOSES OF EXHAUSTION.

- a. Collapse delirium.
- b. Acute confusion (amnesia).
- c. Chronic nervous exhaustion.

#### III. INTOXICATIONS.

1. Acute intoxications.
2. Chronic intoxications.
  - a. Alcoholism (intoxication of alcohol, pathological intoxication, chronic alcoholism and alcoholic degeneration, delirium tremens, Korsakoff's psychosis or chronic delirium, hallucinatory delirious states of alcoholic hallucinosis, acute and subacute forms, and systematized delirium, hallucinatory dementia or alcoholic paranoia, delirious of jealousy, alcoholic pseudoparalysia).
  - b. Morphinism.
  - c. Cocainism.

#### IV. THYROIDogenic INSANITY.

- a. Myxoedematous insanity.
- b. Cretinism.

#### V. DEMENTIA PRAECOX.

Hebephrenic, catatonic, and paranoid forms.

#### VI. PARALYTIC DEMENTIA.

#### VII. INSANITY WITH CEREBRAL DISEASE (cortical gliosis, diffuse sclerosis, Huntington's chorea, multiple sclerosis, syphilis, tabetic psychoses, arterio-sclerotic atrophy, circumscribed lesions—tumors, abscesses, haemorrhages, cysts, atrophies, and traumatic disorders).

#### VIII. PSYCHOSES OF THE PERIOD OF INVOLUTION.

- a. Melancholia (simple and hypochondriacal forms, depressive delirious states, various melancholia).
- b. Possible persecutory delirious states.
- c. Senile dementia (senile dementia, depression, delirium, paranoid states).

#### IX. MANIC-DEPRESSIVE INSANITY (manic and delirious states, depressive and manic states, and mixed conditions).

#### X. PARANOID (of persecution, of grandeur, erotic, and hallucinatory forms, original paranoia, and paranoia of paralytics).

#### XI. EPILEPTIC INSANITY (chronic, periodical attacks, delirious states, states of obsessions, stupor, various delirium, and dipsomania).



## XII. PSYCHOGENIC NEUROSES.

- a. Hysterical insanity (hypocondriacal disorders, change of character, dream states and deliria, moods and states of agitation).
- b. Neurones of fright.
- c. Neurones of satisfaction.

## XIII. ORIGINAL OR CONSTITUTIONAL CONDITIONS.

- a. Nervosity (including congenital neurosthenia).
- b. Constitutional depression.
- c. Constitutional excitement.
- d. Imperative psychoses ("manias" and phobias).
- e. Impulsive insanity (kleptomania, etc.).
- f. Sexual psychopathia.

## XIV. PSYCHOPATHIC PERSONALITIES.

- a. The born criminal, and moral insanity.
- b. The unstable (including pseudo-dipsomania and habitual criminals).
- c. The morbid liars and frauds.
- d. The pseudopsychotics.

## XV. STATES OF DEFICIENT DEVELOPMENT.

- a. Imbecility.
- b. Idiocy.

To any but the expert and special student some of these classifications must, indeed, be mystifying and incomprehensible. They are forbidding to the ordinary student and to the general practitioner, and might well induce him to shun the realm of psychiatry which open before him so uninvitingly and present such obstacles to his progress. And the fact is that they are interesting to the specialist alone because they are as yet quite impracticable from the standpoint of actual utility, as is evidenced by the employment even by the physicians of asylums, who are nothing if not practical alienists, of far simpler schemes of classification in the preparation of statistics for their annual reports and in the histories entered upon their case-books. If the asylum practitioners are compelled for practical purposes to adopt a simple method of classification, how is the novice in psychiatric learning to surpass them in the diagnosis and grouping of his cases? Here, for instance, are two of the latest classifications made for the asylums of New York State by the State Commission in Lunacy, assisted by the director of the Pathological Institute and a committee of superintendents of the hospitals (1905).

The first of these tables is one for statistical purposes only, it having been found to be more practical to separate the statistical classification from our record entirely to clinical-pathological purposes:

## STATISTICAL TABLE OF NEW YORK STATE HOSPITALS.

1. Alcoholic insanity.
2. General paresis.
3. Senile insanity.
4. Epilepsy with insanity.
5. Imbecility and idiocy with insanity.
6. Other psychoses.
7. Not insane.

## CLINICO-PATHOLOGICAL CLASSIFICATION OF NEW YORK STATE HOSPITALS.

1. Brain tumor.
2. Traumatic psychosis.
3. Psychoses accompanying other nervous diseases.
4. Senile psychosis.
5. General paralysis.
6. Alcoholic psychosis (with subdivisions into types).
7. Morphinism and cocaineism, etc.
8. Infective-exhaustive psychoses (dementia type).
9. Allied disorders.
10. Depression not sufficiently distinguished.
11. Melancholia symptomatic.
12. Depressive hallucinosis.
13. Involution melancholia.
14. Disorders allied to the depressions.
15. Parasitic conditions.
16. Dementia praecox.
17. Allied disorders.
18. Manic-depressive psychoses (first, second, third, fourth, etc., attack).
19. Allied disorders.
20. Constitutional inferiority.
21. Hysterical insanity.
22. Epileptic insanity.
23. Insanity and idiocy with insanity.
24. Not classified.
25. Not insane.

In the above, the item 9 implies disorders akin in type, but not sufficiently in etiology, to the toxic and infective-exhaustive processes. Item 14 covers disorders allied to the depressions (Nos. 10 to 13). Item 17 contains the disorders akin to parasitic conditions and dementia praecox.

This latter scheme of classification, developed under the Krapelinian influence, has been further slightly modified with time, as evidenced by the following table of admissions to the New York State hospitals for the year ending September 30, 1909:

Psychosis.	Males.	Females.	Total.
With brain tumor . . . . .	4	2	6
Traumatic . . . . .	17	5	22
Senile . . . . .	270	327	606
Dementic paralysis . . . . .	485	123	608
With other brain or nervous disease . . . . .	169	71	240
Alcoholic . . . . .	455	128	583
Drug and other toxic . . . . .	8	16	24
Infective-exhaustive and syphilitic . . . . .	44	34	138
Allied to infective-exhaustive . . . . .	6	27	33
Symptomatic depressions . . . . .	6	7	13
Depressive hallucinosis . . . . .	17	29	46
Involution melancholia . . . . .	68	139	207
Depression insufficiently distinguished . . . . .	58	73	131
Dementia praecox . . . . .	543	489	1032
Allied to dementia praecox . . . . .	69	80	149
Parasitic conditions . . . . .	94	154	248
Manic-depressive . . . . .	161	241	402
Allied to manic-depressive . . . . .	58	114	172
Epileptic . . . . .	84	64	148
Hysterical, psychasthenic, and neurasthenic . . . . .	14	30	44
Other constitutional disorders and inferiorities . . . . .	31	51	122
Idiocy and idiocy with insanity . . . . .	85	36	121
Unclassified . . . . .	62	64	126
Not insane . . . . .	35	29	64
Total . . . . .	2780	2442	5222



I have several reasons for reprinting this instructive table from the latest available report of the State Lunacy Commission. In the first place, it bears little resemblance to any of the classifications of the English, French, or German schools printed above, even to that of Knapelin, which is its main foundation, and well illustrates the futility of any classification at all in the present state of our knowledge of the pathology of insanity. The diagnoses of these 5149 cases of insanity have been made by the practical alienists in our asylums during the year stated, and it is fair to assume that they had an average of six months for the observation and study of each patient recorded in these statistics. If we add to the cases in the list that could not be classified at all the cases that are put under headings that are ill defined, hazy, confusing, and not clear-cut syndromes, we have the following table:

Unclassified	111
With other brain or nervous diseases	198
Allied to infective-exhaustive	22
Symptomatic depression	12
Depressions undifferentiated	132
Allied to dementia precox	149
Paranoid conditions	248
Allied to manic-depressive	172
Hysterical, psychasthenic, and neurasthenic psychoses	44
Other constitutional disorders and infirmities	122
Total	1210

Now, subtracting from the total admissions the 73 patients that had been committed that year and were found not to be insane, we have nearly 24 per cent. of the cases in which experts, after an average observation for six months, were unable to arrive at a more definite diagnosis of the form of insanity than is shown in the above table. In about one case in eight they could not tell whether a case was true dementia precox or similar to it or allied to dementia precox. In nearly one case in three they could not decide whether a case was true manic-depressive insanity, or somewhat like it, allied to manic-depressive insanity. In about one in five of the infective-exhaustive psychoses there was the same insecurity of diagnosis. This is not recited in criticism of the methods of classification in vogue, for I believe that no better grouping of cases could be made anywhere than has been done here, but is simply brought forward to illustrate the extraordinary difficulty of making a diagnosis and classification of the forms of insanity in a large proportion of cases. If the asylum experts fail so lamentably, with months for investigation of their cases, the general practitioner need not feel discouraged if he is unable to make a correct diagnosis after seeing a patient once or twice, or if later any diagnosis of his should prove to be incorrect. If he calls a case by the old term melancholia, he will be forgiven if later it should find a place among the finer but more difficult distinctions of the following group from the above statistical table:

Symptomatic-depressions.  
Depressive hallucinations.  
Exhaustion melancholia.  
Depressions undifferentiated.  
Manic-depressive.  
Allied to manic-depressive.

Since this book was first published there has been much change in the views of the psychiatric world. The immense strides in general medical science have had their influence also in this department of medicine. There has been accumulated a vast amount of clinical material, much more thoroughly studied than ever before. Our knowledge of clinical forms and phases, of course and outcome, of neuropathology, and of psychology has been enormously advanced. In each new edition of this volume some changes commensurate with this progress have been made, and in this particular edition the classification is radically rearranged in conformity with present-day judgment; at the same time the needs of the student and practitioner are chiefly considered, since the book is written for them and not for the specialist.

Since the alcoholic and other toxic psychoses and the infection-exhaustion psychoses are described under special headings in the chapter on General Etiology, only the following most important types of insanity will be treated in separate chapters:

1. Manic-depressive insanity.
  - a. Manic phase.
  - b. Depressive phase.
  - c. Circular insanity.
  - d. Irregular melancholia.
2. Dementia praecox.
3. Senile dementia and other senile psychoses.
4. General paresis.
5. Paranoia.
6. Neuro-psychoses, hysterical insanity, epileptic insanity.
7. Idiotcy, imbecility, and feeble-mindedness.



## CHAPTER II.

## GENERAL ETIOLOGY OF INSANITY.

THE proportion of the insane to normal individuals may be stated to be about 1 to 300 of the population, though this proportion varies somewhat within narrow limits among different races and countries. It is probable that the intemperate use of alcohol and drugs, the spreading of syphilis, and the overstimulation in many directions of modern civilization have determined an increase difficult to estimate, but nevertheless palpable, of insanity in the present century as compared with past centuries.

The amount of such increase might easily seem to be large, on superficial examination, because of the imperfection of census-taking in the past, the accumulation of the chronic insane, and in new communities the constant upbuilding of new asylums.

**Sex.**—As regards sex, women and men are about equally affected, for the particular etiological factors determining insanity in the one (such as the puerperal period, the menopause, etc.) are evenly balanced by the special causes acting upon the other (struggle for existence, drunkenness, syphilis, etc.), and both sexes are about alike in their susceptibility to the two great etiological elements in alienation of the mind—heredity and mental or bodily strain.

**Age.**—The question of age is of great importance in a study of the etiology of insanity. While individuals are liable to mental aberration at any age, yet there are particular periods of life characterized by special vulnerability. In general, it may be said that this vulnerability is greatest in women between the ages of twenty-five and thirty-five, and in men between twenty and fifty, for it is at middle age that we find the maximum accumulation of etiological factors. But there are physiological epochs that influence markedly the line of psychic morbidity, and these are the periods of puberty and adolescence (fourteen to twenty years), that of genital involution in women (forty-five), and that of senile involution (sixty to seventy years).

But the chief factors in the causation of insanity may be summed up in two words—heredity and strain. The former is responsible for instability of the nervous system, the latter is multifarious in character, comprising all of the stresses, physical and mental, direct and indirect, unobtrusive and environmental, which may undermine the nervous constitution and bring it to its point of collapse.

**Heredity.**—In determining the factor of heredity we must not be content with ascertaining the existence of psychoses in the ascendants, but must seek, by careful interrogation of various members of the family, for some of the hereditary equivalents, such as epilepsy, chorea, hysteria, neurasthenia, somnambulism, migraine, organic diseases of the central nervous system, criminal tendencies, eccentricities of character, drunkenness, etc., for these equivalents are interchangeable from one

generation to another, and are simply evidences of instability of the nervous system. It is the unstable nervous organization that is inherited, not a particular neurosis or psychosis, and it must be our aim in the investigation of the progenitors to discover the evidence of this.

That the statistics of insanity as regards heredity are often feebly gathered is too well known. In the first place, the recorder of the history of a patient frequently neglects to extend his inquiry far enough to include all of the transmissible psychoneuroses, and, in the second, the relatives are prone to conceal any supposed hereditary taint in the family. Here, for example, is a table prepared by the Lunacy Commissioners, showing the causes of insanity in 136,478 admissions to asylums in England and Wales, in which I find the item "hereditary influence ascertained" 20.5 per cent. Surely, so small a figure does not represent the true proportion of heredity as an etiological factor!

It will take many decades of much more careful compilation of histories to establish the actual ratio, but we shall attain nearer to the facts year by year.

No one has better formulated the principles of heredity in relation to insanity than Mercier,<sup>1</sup> who points out, among other things, that, besides the importance of the direct transmission of an unstable nervous system, there is another law of heredity, which is known as the law of singularity. Two parents may be perfectly stable and have normal organisms, and yet produce offspring with unstable and abnormal nervous constitutions, because of the unsuitability of the sexual elements of the parents to each other. The perfect organization of the progeny is the result of three factors—the quality of the germ (which brings matter), the quality of the sperm (which brings force), and the suitability of the one to the other.

The laws of heredity as they relate to insanity may be summarized briefly as follows:

1. The child tends to inherit every attribute of both parents.
2. Contradictory attributes can not be inherited from both parents.
3. The child may inherit the attributes of either parent *solely*.
4. It may inherit the qualities of one parent in some respects and of the other in other respects.
5. It may inherit the father's attributes for one period of existence and the mother's for another.
6. Some attributes have the quality of prepotency, or the tendency to push aside or override other attributes.
7. Attributes which are similar in both parents tend to become prepotent, giving rise to convergent or cumulative heredity.
8. Attributes may be transmitted in latent form from one generation to another, to reappear in a third or fourth or still more remote generation—a phenomenon termed "reversion."
9. Attributes tend to appear in the progeny about the same time of life at which they became manifest in the parents.

<sup>1</sup> "Sanity and Insanity."



10. Attributes of the father tend to be inherited by the sons and of the mother by the daughters.

A study of the above laws will explain many of the puzzling features of psychopathic heredity,—why, for instance, often only a few of the children of a neurotic parent suffer from neuroses or psychoses, and why psychoneuroses may develop in the progeny of healthy parents (latency). It must be remembered, too, that there is a variation in the degree of hereditary taint originated by the several heritable equivalents. Thus, simple acromioclavicula, eccentricity of character, and a preperal or senile psychosis are not so serious a heritage as epilepsy, chronic alcoholism, paranoia, and imbecility. The taint in a family is greater the larger the number of members and branches afflicted. When the degree of hereditary taint is marked, the psychoses which may develop tend to be modified from the ordinary types of such psychoses, and this deviation is termed hereditary degenerative modification,—or, in short, hereditary degeneracy,—while the insanity evolved is designated as a degenerative psychosis. The particular degenerative psychoses are such forms as idiosy, imbecility, feeble-mindedness, periodical and circular insanity, hysterical insanity, acute simple paranoia, polymorphic insanity, etc. A polymorphic course is particularly characteristic of psychic degeneracy, so that sometimes a perfect chain of psychopathic conditions and psychoses will be manifested throughout the life of the degenerate.

The polymorphism of hereditary transmission sometimes manifests itself in what is known as progressive hereditary degeneracy. For example, drunkenness in one generation may lead to simple psychoses in the next, to complex degenerative psychoses, epilepsy, etc., in the third generation, and finally, in the fourth, to idiosy, sterility, and the annihilation of the stock.

It is only lately that the laws of heredity, as they relate to insanity, have begun to be studied in the light of the new theories of Mendel and others. The results of observations of plants have certainly revealed some remarkable facts, but how far these can be applied to the infinitely complex conditions of the human organism and human society remains to be determined by years of most careful investigation.

The indications of degeneracy in an individual are termed the *stigmata* of degeneration, or *stigmata hereditatis*. They may be defined as anatomical or functional deviations from the normal, which in themselves are usually of little importance as regards the existence of an organism, but are characteristic of a marked or latent neurogenic disposition. Much study has of late years been devoted to these indices by many investigators, particularly in their relation to insanity, idiosy, and criminal anthropology, and it behooves all who have to do with the development and care of the human body in any particular—and this refers especially to men of the medical and allied professions—to familiarize themselves with these signs of degeneration, in so far as they concern their own special provinces of work. These stigmata are views

of functional and organic evolution. The deviations from the normal may be in the way of excesses or arrest of development. They must be distinguished from the deficiencies or deformities produced by accidents at birth or by disease. I have said that these stigmata are anatomical and functional, but it is more convenient to divide the functional group into physiological and psychic classes. It is the latter which we are more apt to observe in our social relations with degenerate individuals. The psychic stigmata are always characterized by want of balance or lack of proportion between certain undeveloped or excessively developed faculties and other faculties which are normal. Defect of moral sense, of attention, of memory, will, judgment, or unbalanced excess of musical or mathematical aptitudes may be cited as instances of psychic stigmata. Hence the three following divisions may be made of all the degenerative indices: (1) Anatomical stigmata; (2) physiological stigmata; (3) psychic stigmata.

#### ANATOMICAL STIGMATA.

- Cranial anomalies.
- Facial asymmetry.
- Deformities of the palate.
- Dental anomalies.
- Anomalies of the tongue and lips.
- Anomalies of the nose.
- Anomalies of the eye:
  - Flecks on the iris, iridism, chromatic asymmetry of the iris;
  - narrow pupillary fissures.
- Albinism.
- Concave corneata.
- Micropthalmos.
- Pigmentary pointitis.
- Muscular insufficiency.
- Anomalies of the ear.
- Anomalies of the hand:
  - Polylactyl.
  - Synlactyl.
  - Ectrolactyl.
  - Synolus.
  - Ectroclaus.
  - Phicoclaus.
  - Excessive length of the arm.
- Anomalies of the body in general:
  - Hermin.
  - Malformation of the breasts, thorax.
  - Dwarfism.
  - Gigantism.
  - Infantilism.
  - Feminism.
  - Masculinism.
  - Spina bifida.
- Anomalies of the genital organs.
- Anomalies of the skin:
  - Polysmia.
  - Hypertrichosis.
  - Absence of hair.
  - Premature grayness.



## PHYSIOLOGICAL STIGMATA.

- Anomalies of motor function:
  - Retardation of learning to walk.
  - Tics.
  - Tremors.
  - Epilepsy.
  - Nystagmus.
- Anomalies of sensory function:
  - Deaf-muteness.
  - Neuralgia.
  - Migraine.
  - Hyperesthesia.
  - Anesthesia.
  - Blindness.
  - Myopia.
  - Hypermetropia.
  - Astigmatism.
  - Dilatation.
  - Hemianopia.
  - Concentric limitation of the visual field.
- Anomalies of speech:
  - Mutism.
  - Defective speech.
  - Stammering.
  - Stuttering.
- Anomalies of genito-urinary function:
  - Sexual irritability.
  - Impotence.
  - Sterility.
  - Urinary incontinence.
- Anomalies of instinct or appetite:
  - Uncontrollable appetite (food, liquor, drugs).
  - Mercenism.
- Diminished resistance against external influences and diseases.
- Retardation of puberty.

## PSYCHIC STIGMATA.

- Idiocy.
- Imbecy.
- Imbecility.
- Feeble-mindedness.
- Pavor nocturnus.
- Proecy; one-sided talents; disqualification.
- Eccentricity.
- Moral delinquency.
- Sexual perversion.

Having made this attempt to classify the various stigmata, we may now proceed to examine them in some detail:

**Cranial Anomalies.**—The most important features to be noted in connection with the head are asymmetry and a variety of deformities. It is necessary to an understanding of these stigmata to go over briefly a few facts of craniometry and cephalometry.

A score or more of distinguished anthropologists of the present century have been trying to discover racial distinction in human skulls; but the fact is that there are not so many characteristics of race in the cranium as in other parts of the body, and, accordingly, there are still wide differences of opinion as regards a scientific craniological classification. Races have been mingling so many thousands of years that

cranial dissimilarities are the rule among them, even in tribes, and to some extent in families. These diversities of form have been designated as *delichcephalic*, *mesocephalic*, and *brachycephalic*—words which merely convey an idea of the relation of the length to the breadth of the skull when viewed from above. The anteroposterior is to the biparietal diameter as 100 is to  $x$ , is the formula for determining this "cephalic index." All length-breadth indices below 78 are considered *delichcephalic*; from 78 to 80, *mesocephalic*; and above 80, *brachycephalic*. We may assume that the physiological limits of this index are 70 to 90. This is based upon thousands of measurements of skulls



Fig. 262.

by various investigators. Any excess or diminution of these figures must hence be regarded as pathological (Fig. 269).

But while one skull may be narrower or broader than another, there is compensation in other diameters. The *delichcephalic* has a greater vertical diameter, for instance, than the *brachycephalic* skull.

Besides these characteristics, something may be said regarding the physiological asymmetry of the skull. The fact that the arms and hands are not symmetrical on the two sides of the body, either in size or function; that the legs and feet are not symmetrical; that the left cerebral hemisphere is larger and more complicated than the right, would naturally lead us to anticipate some slight asymmetry of the two sides of the skull, and the facts of observation support us in the statement that asymmetry is the rule and perfect symmetry the exception. More than a thousand postmortem examinations, the examination of several hundred heads, and an inspection of some collections of skulls, such as that of Blumenbach, where I have particularly noted this point, together with the testimony of others, justify me in this assumption.

Asymmetry sometimes reaches extraordinary proportions,—often with quite a normal state of brain function, often with marked psychopathic changes. Outside of purely physiological asymmetry, we have that depending upon defective development and disease. One of the first of nature's constructive principles in fashioning the skull is the struggle of its contents for volume. Hence, as long ago pointed out by Virchow, premature synostosis of any cranial suture will lead to compen-



satory deformity. So, too, will arrest of development in any center of



Fig. 268.—Chemocephalus.

ossification, or a unilateral aphasia or hyperplasia of the skull bones, or of the contents of the skull.

Aside from the deformities of the head which are congenital in character, the diseases which most commonly produce cephalic deformation in early life are rickets and hydrocephalus; in later life, tumors, exostoses, etc.; while at all periods of life the shape of the skull is metcured by injuries, from a forceps delivery to a falling brick. The following are some of the commoner designations of well-known cranial deformities:

**Chemocephalus** is flat-headedness. In this there is flatness at the

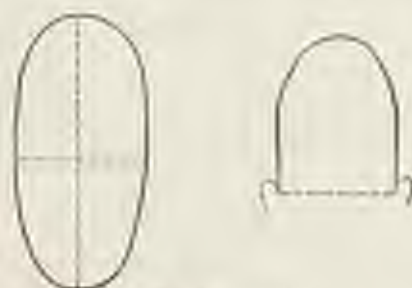


Fig. 270.—Leptocephalus.

top of the head. The condition is also called **platycephalus** (Fig. 270).

**Leptocephalus**.—Early synostosis of the frontal and sphenoid produces **leptocephalus**, or narrow-headedness (Fig. 271).

**Macrocephalus** is a large head, usually due to hydrocephalus.

**Microcephalus** is a small head, due either to aplasia of the brain or premature synostosis of the sutures (rarely the latter).

**Oxycephalus**, or steeple-shaped skull, is due to synostosis of the parietal with the occipital and temporal bones, with compensatory development in the region of the bregma. Another name for this is **oxycephalus** (Fig. 272).

**Plagiocephalus**, or oblique deformity of the head, is due to unilateral synostosis of the frontal with one of the parietal bones (Fig. 273).

**Scaphocephalus** is probably caused either by too early union of the sagittal suture or by the development of both parietal bones from one center. The top of the head is keel-shaped (Fig. 274).

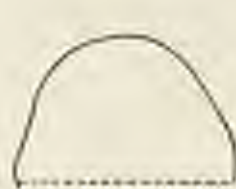


Fig. 274.—Scaphocephalus.

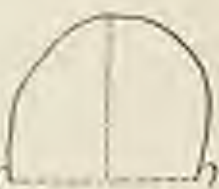


Fig. 275.—Trigonocephalus.

**Trigonocephalus**.—Premature union of the frontal suture, resulting in very narrow forehead and great width behind, giving rise to the term trigonocephalus (Fig. 275).

The two systems of measurement—the craniometrical and the cephalometrical—differ but slightly from each other, the former, of course, being the more exact, since every portion of the naked skull is attainable.

I would recommend the following series of measurements to be



Fig. 276.—Mesencephalus.

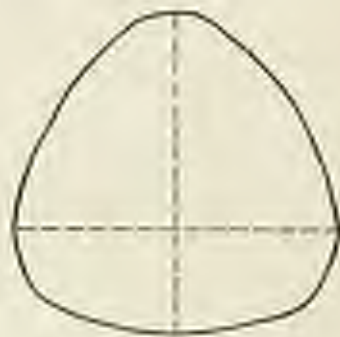


Fig. 277.—Trigonocephalus.

taken—eleven in number—in order to form a just idea of the capacity, shape, and symmetry of any head (Figs. 276 and 277): (1) The circumference; (2) the naso-occipital arc (*N* to *T*); (3) the nasobregmatic arc (*N* to *B*); (4) the bregmatolambdoid arc (*B* to *L*); (5) the biauricular arc; (6) the anteroposterior diameter (*S* to *O*); (7) the greatest transverse diameter (length-breadth index); (8) the biauricular diameter; (9) the two auriculobregmatic radii; (10) the facial length; (11) the empirical greatest height (*B* to *P*).

In addition to acquiring these mathematical data, ophthalmoscopic



drawings are invaluable as exhibiting deformity clearly to the eye. Hence, the horizontal circumference, meso-occipital curve, and bimastoid curves should be taken with a strip of lead, or, what is better, with the instrument devised by Lays (on the principle of the latter's conformateurs), and the curves projected on paper.

Dolichocephalic heads, as a rule, have narrow, and brachycephalic have broad, faces. Something should here be said concerning prognathism, of which there are several forms. The best method of determining it is to measure the angle made by a line drawn from the nasal root to the junction of the inferior nasal spine and alveolar process (Fig. 277, *N* to *x*) with a vertical line (dropped from the nasal root to Broca's horizontal). It is found that every normal skull exhibits this subnasal prognathism, but there is a wide variation in degree. Extrasubnasal prognathism, orthognathism, and opisthognathism—meaning extreme projection, straightness, or inclination backward of the subnasal line—are pathological.

The empirical greatest height of the head is an approximate measure-



FIG. 276.



FIG. 277.

ment of the distance between the basion and vertex of the skull (*B* to *β*, or *U*). A line from the external occipital protuberance to the lowest median point of the superior maxilla, just above the incisors (*T* to *M*), passes almost directly through the basion. Hence, in cephalometry, by taking this diameter and the radii from each extremity to the bregma, we have a triangle (*M, β, T*) whose height (*B, β*) is easily ascertained. The height averages 13.3 cm. in men, 12.3 in women, and the physiological variation is from 11.5 to 15.

The only instruments necessary for obtaining the data just described are a pair of calipers, the tape-line, and a strip of sheet-lead two feet long by  $\frac{1}{4}$  or  $\frac{1}{2}$  of an inch wide. Benedikt's calipers (manufactured by Wolters in Vienna), which are here illustrated, are to be recommended for their exactness (Fig. 278), as are also those that I have had made for my own use (Fig. 279).

Excessive prognathism is found among criminals, in microcephali, and in cases of hemi- and paraplegia spastica infantilis. Skulls known as crania prognatha have considerable pathological significance. In these, lower teeth project beyond the upper, and the inferior maxillary

angle is obtuse, due, probably, to aplasia of the upper or hyperplasia of the lower maxilla.

The demonstration of the empirical greatest height is often quite valuable as an index of degenerative and neuropathic types. The following are some general points which should be considered in the examination of these cases:

A skull below the normal type in volume belongs to an abnormal individual.

Undertypical measurements of the head should always lead us to entertain the suspicion of defective cerebration.

Abnormal smallness of any part of the skull permits the conclusion that the part of the brain in its neighborhood is imperfectly developed.

Excessive development of the head has a double signification. It is



FIG. 278.—Brodsky's calipers.

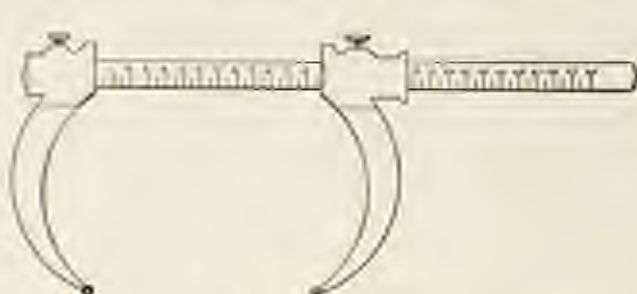


FIG. 279.—Andrew's calipers.

always pathological, but may mean abnormality of brain or successful compensation. Wormian bones are also doubly significant. They either represent a pathological process or a successful effort of nature in repair.

Hemiplegia spastica infantilis, epilepsy, and intellectual or ethnic weakness often exhibit unilateral aplasia of the skull.

The skull is representative of the brain only during the years of its development, and it must be remembered that psychopathic deterioration often has its inception subsequent to the completion of the process, when no impression can be made upon its bony walls.

I have prepared a table of the measurements recommended, showing the averages in adults, male and female, together with the physiological variation, excesses above or below which are significant of mortality. It is based upon the examination of some hundreds of skulls and heads, and upon statistics given by various authorities who have made especial study of this department of anthropometry. Hence it may be depended upon as a fair estimate of the dimensions of the head in most of the Caucasian races. The table is as follows:



TABLE 19. CRANOMETRICAL MEASUREMENTS.

	AVERAGE IN Adults IN Centimeters.		Differences in Variation.	REMARKS.
	Males.	Females.		
1. Circumference, . . . . .	52	50	48.5-57.4	Roughly approximated, the volume is to the circumference as 1:250 c.c. is to 50 cm.
2. Volume, . . . . .	1500	1300	1300-1750	
3. Naso-occipital arc, . . . .	32	31	28-34	In figure, <i>N</i> to <i>T</i> .
4. Naso-bregmatic arc, . . . .	12.5	12	10.0-14.0	<i>N</i> to <i>B</i> .
5. Regnato-lambdoid arc, . . . .	12.5	11.9	9.1-14.5	<i>B</i> to <i>L</i> .
6. Hilar-auricular arc, . . . .	32	31	28.6-35	
7. Anteroposterior diameter, . .	17.7	17.2	16.5-19	<i>X</i> is 0.
8. Greatest transverse diameter	14.6	14	12-16.5	The formula for the length-breadth index is: Length : Breadth :: 100 : <i>x</i> . An index below 78 is dolichocephalic : 78 to 80, mesocephalic : above 80, brachycephalic.
9. Length-breadth index, . . . .	82.0	82.8	76.4-87	
10. Biquadrilateral diameter, . .	12.4	11.9	10.9-13.9	 The height <i>N-X</i> of the triangle <i>E, B, F</i> formed by the antero-subheptagonal radii and the biquadrilateral diameter, averages 11.17 with a variation from 10 to 12.05.
11. Anterobregmatic radii, . . . .				
12. Facial length, . . . . .	12.57	—	10.5-14.4	From nose of nose, <i>N</i> , to lowest part of chin.
13. Empirical greatest height, . .	13.3	12.5	11.5-15	The empirical greatest height, <i>E, F</i> , is obtained by measuring the sides of the triangle <i>N, L, T</i> .

These measurements are those of the adult human skull. As the hair and scalp averaged about 2 cm., about 6 per cent. should be deducted in the head measurements Nos. 1, 3, and 6 to obtain those of the skull. In taking the diameters Nos. 7 and 8, deduct 1 cm. (the scalp averaging 3 mm. in thickness), and from the shorter radii, such as Nos. 10 and 11, subtract but 7 mm.

**Facial Asymmetry.**—Inequality of the two sides of the face—when congenital and not due to some such disease as hemiatrophy—is to be looked upon as a stigma of degeneration. In the same category may be grouped various irregularities, and such conditions as excessive prognathism or retrognathism. Great prominence or unequal promi-

nence of the malar bones is to be observed, and also asymmetry of the orbits (Fig. 280).

**Deformities of the Palate.**—In connection with the soft palate, deformation of the uvula may be mentioned. As regards the hard palate, I have dwelt upon its deformities at some length in an article in the "*International Dental Journal*" (December, 1895), and the facts there brought forward may be recapitulated as follows:

While the palate occupies but a small place in this great category of hereditary stigmata of all kinds, it is one of the anatomical group, and this group is for many reasons the one of greatest importance. In this



Fig. 280.—Osteopsychosis, aged forty years, with glabrous face and skin and facial asymmetry.

group, too, it occupies a distinctive place as being among the most striking, frequent, and significant of the anomalies.

The arch of the hard palate presents considerable variation within strictly normal anatomical limits. A large, wide, moderately high vault is that may be called a normal standard. It means the highest evolution, judging from the fact that the mouth-cavity increases in capacity as we ascend the vertebrate series. Deviations from that standard are not at all infrequent, and yet such deviations may be normal. Thus, the palate may be low and broad, or it may be high and narrow; it may be short or long in its anteroposterior diameter; it may be ridged unduly along the palatine sutures, or it may present marked rugosities on its surface, especially in the anterior region; yet these variations are normal. Probably we may look upon these pos-



larities as a species of compensatory development. Just as in a study of heads we find some very long and low, and others short and round and high, and recognize the fact that the shortness in one dimension is compensated for by a corresponding increase in another, so we may regard variation in palatine diameters.

The pathological palate has not been studied as much as it deserves to be. Save occasional and casual references to the "Gothic" palate in literature, and one or two papers upon the "*torus palatinus*," very little has been written upon the subject. In my paper, previously referred to, I have attempted to classify such pathological palates as could be justly looked upon as indicative of degeneracy. The word Gothic having been so long in use, and the hard palate being much like an arch or roof,<sup>1</sup> I have followed architectural nomenclature in the classification offered.

#### **PATHOLOGICAL PALATES:**

1. Palate with Gothic arch (Fig. 281).
2. Palate with horseshoe arch (Fig. 282).
3. The dome-shaped palate (Fig. 283).
4. The flat-roofed palate (Fig. 284).
5. The hip-roofed palate (Fig. 285).
6. The asymmetrical palate (Fig. 286).
7. The *torus palatinus* (Fig. 287).

The seven varieties named are to be looked upon as types merely. Each type will be found to present variations and combinations with other forms. Thus, the Gothic arch may have a low or high pitch and be short or long. The horseshoe arch (a familiar one in Moorish architecture) is always easily distinguished, but, owing to its conformation, a cast can not well be taken of it to show it in a perfect outline. The dome-shaped palate may be high or low, may be combined with asymmetry or *torus*. The presence of a *torus* in the Gothic variety is apt to destroy the purely Gothic form, and may cause it to resemble the flat-roofed palate. Under the heading of flat-roofed palate I should include all such palates as are nearly horizontal in outline, as well as those with inclined-roof sides but flattened gable. In the hip-roofed palate we have the sloping sides as usual, but also a marked pitch of the palate roof in front and behind; occasionally one may meet with a palate of this kind with so remarkable a pitch from before backward that it is almost like a Gothic roof turned about so that the gable runs transversely.

Asymmetry in the palate is commonly observed in many of the previously described forms, but occasionally is the only noteworthy peculiarity. It is usual to find asymmetry of the face and skull in cases with an asymmetrical palate. The *torus palatinus* (Latin *torus*, swelling) was first mentioned by Chassaignac as a mediopalatine exostosis. It is a projecting ridge or swelling along the palatine suture, sometimes in its whole length, sometimes in only a portion of its course. It is always congenital. It varies considerably in its shape and size, so

<sup>1</sup> There is some confusion in literature of the roof of the mouth, or hard palate, referred to in this paper, with the dental arch, which is quite another thing.

that as many as five or six different species of *toeni* are recognized. It may be wedge-shaped, narrow, broad, very prominent, or irregular. I have said nothing about cleft-palate, for I am not sure that it may be



Fig. 881. — Palate with Gothic arch.



Fig. 882. — Palate with horseshoe arch.

classed among the well-marked stigmata of degeneration. I have found but two or three cleft-palates among the 450 idiots and imbeciles on Randall's Island, while a number of cases of this kind with which I



have come in contact in my professional life were very far from degenerates. However, it would seem that there is great need of a faithful study of a large number of cases of cleft-palate in relation to the ques-



Fig. 282.—The clove-shaped palate.



Fig. 283.—The bat-eared palate.

tion of degeneracy. The deformed palate is, to my mind, one of the chief anatomical stigmata of degeneration.

It is true that, from this single indication, it would not be strictly

scientific to adjudge an individual a degenerate. Occasionally, perhaps, a case presents itself where this anatomical stigma alone would suffice to insure a diagnosis of this nature; but usually other stigmata coexist, such as cranial anomalies, deformities of the ear, and the like. The



Fig. 285.—The bip-costal palate.



Fig. 286.—The asymmetrical palate.

frequency of the pathological palate among marked degenerates, such as the insane, idiots, and epileptics, has been testified to by many investigators. Thus, Talbot reported 43 per cent. of abnormal palates in 1605 inmates in institutions for the feeble-minded. Ireland makes it nearer 50 per cent. Clarou, a later writer than these, found abnor-



and palates in 10 per cent. of apparently normal persons, in 82 per cent. of idiots and feeble-minded, in 76 per cent. of epileptics, in 80 per cent. of cases of insanity in general, in 70 per cent. of the hysterical insane, and in 35 per cent. of cases of general paralysis. Nisko has studied particularly the torus palatinus in 1449 individuals, normal and psychopathic; he found it present in 25.9 per cent. of psychopathic women (insane, epileptic, idiot, and criminal), 32.9 per cent. of epileptic women, 34.4 per cent. of criminal women, 22.7 per cent. of normal women. The percentages were smaller in men than in women. A narrow torus is more common than a broad one.

Stieda examined 1500 skulls for the torus from an anthropological point of view. The skulls were of Prussians, Armenians, Africans, Frenchmen, Russians, and Asiatics. He decided that it has no anthropological significance; gives no racial distinction. While the torus is



Fig. 272.—Torus palatinus (broad, with incisor).

undoubtedly of value as an index of degeneration, particularly where it is well marked, it probably has less importance in this respect than some of the other forms of pathological palate.

**Dental Anomalies.**—Among anomalies of the teeth are microdontism, microdontism, projecting teeth, badly placed or misplaced teeth, double row of teeth, or teeth which are striated transversely or longitudinally. Curves of the teeth and Hutchinson's teeth are due to neglect or disease. The latter, however, may often be considered as a stigma of degeneration. Then there is a retardation of the first and second dentition.

**Anomalies of the Tongue and Lips.**—A very large tongue (macroglossia) is not infrequently observed among the lower classes of degenerates, as in idiocy. Sometimes there is microglossia, asymmetry of the two halves, or bifidity of the point. Harelip is somewhat more common than cleft-palate, but, like the latter, its exact standing as a

degenerative stigma is not fully determined. Undue swelling or puffiness of the lips is noteworthy.

**Anomalies of the Nose.**—Marked deviation of the nose to one side or the other should be noted. Taken alone it may possess little significance, but in conjunction with other stigmata it is of value. The nose may be absent, or present defect of osseous development (*nasus colubinus*) or atresia of the nasal fossæ.

**Anomalies of the Eye.**—The pathological conditions of the eye have been placed in two groups in the foregoing classification, since some are anatomical and some physiological. To enumerate them altogether, they are as follows:

ANATOMICAL.	PHYSIOLOGICAL.
Plecks on the iris.	Blinchness.
Strabismus.	Myopia.
Chromatic asymmetry of the iris.	Hypermetropia.
Narrow palpebral fissures.	Astigmatism.
Albinism.	Daltonism.
Congenital cataracts.	Hemeralopia.
Pigmentary conjunctivitis.	Concentric limitation of the visual field.
Microphthalmos.	Nystagmus.
	Muscular insufficiency.

It is true that any one or two or more of these conditions present do not certainly indicate degeneracy, but they are significant in connection with other abnormal states, and all of them are more frequently observed in degenerate individuals, especially the lower orders, than in normal persons. In idiots, convergent strabismus, due to defect of refraction and in conjunction with hypermetropia, is very common. Muscular insufficiency and nystagmus (lateral or rotatory) are also often met with in this class of cases. In paralytic and other idiots and imbeciles homonymous hemianopsia is sometimes met with.

**Anomalies of the Ear.**—Deformities of the ear have been deservedly well studied, for as stigmata of degeneration they take high rank, like anomalies of the hard palate, in the anatomical group. Mond, Stahl, Wildermuth, Binder, and, more recently, Schwalbe, have given us especially good studies of these conditions. From their writings and my own studies, the following classification (following Binder) into twenty-two varieties may be made:

I. Abnormally implanted ears; they project too far or lie too closely, are placed too high or too low, too far forward or too far backward on the head.

II. Excessively large ears: (1) absolutely too large; (2) relatively too large in small or microcephalic individuals.

III. Ears which are too small.

IV. Too marked conchoidal shape of the ear. The details of the ear (antihelix and crura, etc.) are but slightly marked, while the helix outlines the ear like the rim of a funnel.

V. Ears which have a general ugly shape. The breadth of the upper part may exceed that of the lower, and vice versa; excessive length; ears without lobules; unusually short ears.



VI. Ear not uniform in width; usually a long ear with one or more constrictions in its breadth.

VII. The Blairville ear; asymmetry of various kinds of the two ears. In most cases the asymmetry is due to an anomaly of the left ear.

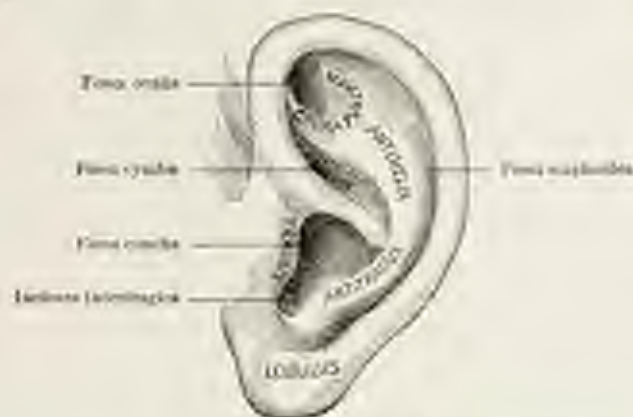


Fig. 183.—Normal ear.

VIII. The ear without lobule; there are usually other deformities of this ear besides the absence of lobule, such as too large a concha, prominence of the anthelix, etc.

IX. The ear with adherent lobule; the lobule is enlarged, adherent, and inclines downward toward the cheek.



Fig. 184.—Blairville ear (also excessive length of ear).



Fig. 185.—Adherent ear.

X. The Stahl ear, No. 1.<sup>1</sup> A series of anomalies of the helix. The helix is broad, like a band, and coalesces with the cartilage

<sup>1</sup> See "Zeitschrift für Psych." vol. xvi.

of the *crura furcata*. The *fossa oralis* and *fossa scaphoidea* are scarcely to be seen. The lower half of the helix is obliterated. There are occasionally slight variations from this type.

XI. The Darwin ear; helix interrupted where its transverse portion passes into the descending, and at this point is a projection of the rim shore and outward, like the pointed ear of lower animals.

XII. The Willernauth ear.<sup>1</sup> The anthelix projects so far as to form the most prominent part of the auricle.

XIII. The ear without anthelix or *crura furcata*.

XIV. The Stahl ear, No. 2. Multiplication of the divisions of the *crura furcata*, so that there are three instead of two *crura*.

XV. Willernauth's Aztec ear. Lobule wanting; the whole ear seems pushed forward and downward; the *crus superius* of the anthelix esplices with the helix, while its *crus anterius* is scarcely perceptible.

XVI. The Stahl ear, No. 3. Only the *crus anterius* of the *crura furcata* is present, while the auricle seems divided into two halves by a ridge from the antitragus.

XVII. The ear with double helix.



Fig. 97.—Stahl ear, No. 1.



Fig. 98.—Darwin ear.

XVIII. The ear with too large or too small a concha.

XIX. The ear with continuous *fossa scaphoidea*. The *fossa* passes down into the lobe.

XX. The Meek ear. A form marked by abnormal development of the helix, anthelix, *fossa scaphoidea*, and *crura furcata*, so that the folds of the ear seem obliterated, and the ear is smooth, larger than usual, often prominent, and with thin edge.

XXI. Ears misshapen by abnormal cartilage development. Here belong all irregular cartilaginous growths and thickenings except those caused by hematomas of the ear.

XXII. Various peculiarities, difficult to classify, are included here, such as abnormalities of the semilunar incisure of the tragus and of the meatus, coloboma of the lobule, hairiness of the different parts of the auricle, necessary ears, clefts, etc.

<sup>1</sup> Warr. *Curios. Natur.*, 1800, No. 40.



The most important malformations of the ear—those that may be regarded as belonging to the stigmata of degeneration, and those, too, which are striking and plain to the eye—are to be summarized as follows:

- The deep position of the *crus auriculae*.
- Marked prominence of the *anthelix*.
- Excessive broadening of the *ear*.
- Stunted development of or absence of the *helix*.
- Trifurcation of the *anthelix*.
- Widening of the *fovea scaphoidea*.
- Absence of the *crus superius*.
- Complete absence of *lobule*.
- Asymmetry of the two ears.
- Excessive enlargement or dilatation of the *ostia*.
- Excessive conchoidal structure of the ear.

Reference is occasionally made in literature to the *Cagot* ear. The *Cagot* is a species of *cretin* in the French and Spanish Pyrenees, in which one of the chief physical deformities is absence of the *lobule* of the ear.

Binder states that the *adherent lobule* exists in almost one-third of normal persons, and in the photographs of several hundred distinguished persons 15 per cent. had abnormal *lobules*. At the same time more



Fig. 393.—Excessive length of nose; facial asymmetry.

than twice as many *adherent lobules* are found in degenerates as in normal individuals.

Now, with regard to statistics of malformed ears in degenerate individuals, Wilmersmith noted this condition in 11 per cent. of 142 idiots. Binder found 64 per cent. of degenerate ears in 354 insane persons. It is to be remarked, however, that Binder was more careful in his examinations, and by long practice had acquired more expert knowledge than Wilmersmith. Fränkel observed degenerate ears in 23 cases out of 32 with *cranium pegmatum*.

Knecht found 20 per cent. of degenerate ears among 1274 criminals, 27 per cent. among 48 epileptics, and 32 per cent. among 84 insane.

Binder noted degenerate ears in 33 persons outside of institutions, supposed to be normal individuals. Inquiring closely into their histories, he discovered that 7 of them had insane parents, brethren, or children; in 19 there were decided psychic abnormalities, and only 7



Fig. 284.—Front, hoodlike helix; no ant. helix; no lobule; excessive size of head (sym.).



Fig. 285.—Excessive length of ear; fusion and dilatation of helix, antihelix, antitragus, and lobule.



Fig. 286.—Triplification of crura helix, well-formed helix and antitragus, almost lobule.



Fig. 287.—Flange in antihelix, slight Darwin tubercle, slight antitragus.



Fig. 288.—No ear tagette; no antihelix; small firm concha; few details of ear.



Fig. 289.—No lobule; no Darwin tubercle; shallow fossa trochlearis; fusion of helix, antihelix, and antitragus; a type of round ear, No. 3.



Fig. 290.—Prominent antihelix; underdeveloped helix; absence of lobule; dilatation of the concha; Wickenburg ear, No. 1.

were apparently normal persons. As regards heredity, it is very common for children to inherit ears with the identical characteristics of those of one or the other parent, but, on the other hand, it is not uncommon for the ears of the children to be quite different.



**Anomalies of the Limbs.**—Paralysis, atrophy, retarded growth, club-foot, and athetosis are conditions due to disease of the brain, and are observed in many cases of paralytic idiosy. These are not properly stigmata of degeneration, although they may be such under some circumstances, as, for instance, when club-foot or club-hand has a teratological origin. On the other hand, there are anomalies having a hereditary character, which are essentially degenerative indices. Among these may be mentioned congenital luxations, supernumerary fingers or toes (polydactyly), fusion of fingers or toes (syndactyly or arachnodactyly), excessive length of the arms as compared with the rest of the body and the lower limbs, missing fingers or toes (retrodactyly), missing limb (ectromelia), fusion of the extremities (symphalangism or symphalangism), or absence of parts of limbs so that they are excessively short (phocomelia). There may also be anomalous brevity of some digits as compared with the relative proportions of normal digits. Excessive volume of limbs (megaloachia) or digits (megaloachyly) or excessive gracility of limbs (oligomelia) or of digits (oligodactyly) also deserve mention.

**Anomalies of the Body in General.**—Local malformations are naturally of more importance than general anomalies of the whole form, but it is necessary to study the relative proportions of the entire figure from an anthropometrical point of view, and to compare the results with normal standards. Excessive diminutiveness of figure, as well as excessive or giant growth, are indications of degeneracy. So, too, are infantile characteristics in an adult, feminine peculiarities in males, and masculine traits in females. In this regard, observations of the relative proportions of the shoulders and pelvis are particularly useful. The occult form of spina bifida with local hypertrichosis is met with. Deviation of the vertebral column among neuropaths is mentioned by Eber. They may be lordoses, scolioses, or kyphoses in various degrees. The coccyx may present peculiarities, such as simulation of a tail. Thoracic asymmetry or other deformity is observed at times. Absence of pectoral muscles, or of muscles in various parts of the body, has significance. Hernia are evidence sometimes of arrest of development of some part of the abdominal wall. Excessive development of mammary glands in males, or their absence or reduplication (polymastia) in either sex, constitutes an evidence of degeneracy.

**Anomalies of the Genital Organs.**—Among the genital anomalies



Fig. 301.—Phocomelia, right arm. In typical case, right forearm several inches shorter than left, same otherwise perfect.

in males are cryptorchidism; unilateral or bilateral microorchidia; spurious hermaphroditism; insufficient development of the entire genital apparatus; hypospadias; epispadias; defect, torsion, or great volume of the prepuce; median fissure of the scrotum; imperforate mentus.

In females the labia may be abnormally large, simulating a scrotum; sometimes very small. The clitoris may be exceedingly large. The labia minora may be hypertrophied. Sometimes there are intermediate folds between the labia minora and labia majora. The labia minora may be pigmented, particularly in brunets and when they are hypertrophied. There may be imperforate vulva, or atresia of the vagina, or double vagina; uterus bicornis is sometimes met with.

Anomalies of the genito-urinary apparatus should always be sought for, for, though most frequent among idiots, imbeciles, epileptics, and the like, they are by no means rare in other classes of degenerates and in degenerate families. In males, defect of the testicles often coincides

with general excess of growth in the whole body or in the lower extremities, such as is often produced by castration in man and lower animals.

#### Anomalies of the Skin.—

Among the anomalies of the skin are to be mentioned adipose thickening; polyactria; proscenisms and often abnormal development of the hairy system; hair along the spinal column; rudimentary tail; premature grayness; a glabrous chin in grown men; persistent lanuginous character of the hair; excessive growth of hair on the chin and breast in women; complete or partial discoloration of the hair (albinism, vitiligo); local or general hypertrichosis; partial or complete absence or fetal state of the nails; melanism of the skin; pigmentary or vascular nevi; molluscum; ichthyosis; vitiligo; albinism; pigmented spots.

**Anomalies of Motor Function.**—Delay in acquiring a knowledge of the proper use of muscles for walking, eating, and the like may often be regarded as an index of degeneracy. Where ordinary etiological factors may be excluded, tremors, tics, epilepsy, and nystagmus may have a similar value. Even when not congenital, they often indicate hereditary instability of the nervous system.

**Anomalies of Sensory Function.**—The numerous anomalies of function in connection with the eye have already been mentioned. Congenital deafness has also its significance. So, too, have hereditary forms of migraine and neuralgia. Certain defects or excesses in general cutaneous sensibility have been noted as frequent among degenerates. Thus several excellent writers on this subject have stated that a



FIG. 302.—Hypertrophism in a female idiot.



general anesthesia is not uncommon, especially among lower classes of degenerates. In some instances there is hyperesthesia.

**Anomalies of Speech.**—It may be questionable as to how far stammering and stuttering are to be looked upon as functional degenerative stigmata, but they are certainly found more often in children with a neuropathic inheritance than in children with good heredity. Delay in the acquisition of language and complete or partial defect of speech have more significance.

**Anomalies of Genito-urinary Function.**—Sexual irritability, impotence, sterility, and urinary incontinence must be considered as indices of neuropathic disposition. Retardation of puberty in both sexes, but especially in the male sex, is a noteworthy indication.

**Anomalies of Instinct or Appetite.**—It has been pointed out that, among all degenerates, there is a taste or appetite for certain foods or drugs which tends to favor their dissipation (alcohol, morphin, cocaine, and the like). In many cases of inebriety the uncontrollable appetite is to be looked upon as a functional stigma of neuropathic inheritance. Gluttony, surfeits, and the like are usually similar indications.

**Miscellaneous.**—A diminished resistance against external influences (such as strains of various kind) and diseases is significant. Great precocity of intellectual development and of certain aptitudes, and morbid emotional conditions, are among suspicious indications of a neuropathic basis.

The psychic stigmata of degeneracy need only the foregoing enumeration.

### PHYSICAL AND MENTAL STRAIN.

At the beginning of this chapter I spoke of the etiology of insanity as being describable in two terms, heredity and strain—heredity, which renders the nervous organization unstable, the strain, which causes the unstable nervous centers to collapse. Doubtless there are limits of endurance in any organization, no matter how strongly balanced. If the strain be great enough, but the instances of insanity developing in individuals with properly balanced and adjusted nervous organizations are rare indeed. The strain which breaks the unstable nervous system is physical or moral, often both. What organism could withstand the assaults upon its integrity of all three of these factors—heredity, physical ill-health, and enervating care? It is difficult to estimate accurately the proportion of one cause as compared with another, since usually several are associated in the same case; but I believe that statistics will support me in the statement that the physical causes (in which I include alcohol, bodily diseases and disorders, accident and injury, old age, the puerperal state, the menopause, and the like) surpass the moral causes (grief, domestic trouble, business worry, overwork, religious excitement, love affairs, fright, nervous shock, etc.) as factors in insanity by about two to one—that is, twice as many are made insane by physical strain as by mental strain. It now behooves us to

examine these diverse stresses, and to show how some of them give a special color or character to the psychosis developed. It is best to present them somewhat in the order of their frequency, under two or three categories, the most common and most important first, the rarest last. The physical, physiological, and moral causes, then, will be considered in the following order:

*Physical:*

1. Toxic (antitetanus, alcohol, narcotics, metallic poisons, etc.).
2. Bodily diseases and disorders (syphilis, acute and chronic diseases of the nervous system).
3. Trauma to the head.
4. Nervous exhaustion.

*Physiological:*

1. Puberty.
2. Puerperal state.
3. Menopause.
4. Senility.

*Moral:*

1. Emotional strain.
2. Irritation.

**Toxic Influences.**—It is not surprising that deleterious agents in the blood, which bathes every cell and fiber of the nervous system, carrying thither the necessary nutritional elements and removing thence the waste products, should readily overstimulate, retard, pervert, or destroy its high functions. Some of these agents (like alcohol) also affect the nutrition of the central nervous system, by inducing disease of the arteries and of the stomach, liver, and kidneys. Some of the poisons cause insanity by long-continued chronic action upon the nervous system, and others by acute intoxication.

**Auto-intoxication.**—Accumulation of deleterious agents in the blood in such quantity as to affect the nervous system—*e. g.*, carbonic acid and the poison of diabetes and of uremia—has been long known to medical science; but the more mysterious poisons produced by disease in various parts of the body, by fermenting or putrefying substances in the alimentary tract, and by some of the acute infectious fevers, have only of late taken an important place in the etiology of the psychoses. We do not yet know how frequently auto-intoxication from absorption of intestinal poisons determines insanity, but the facts thus far collected point to the origin of a considerable number of cases from this cause. These cases are usually of depressed type, but sometimes maniacal.

**Alcohol.**—While the position of autotoxemia as a factor in etiology is not yet determined, we may say of alcohol that it stands foremost (after heredity) as a single, independent cause (eighteen to twenty per cent. in males). Acute alcoholism rarely induces a psychosis. Alcoholic insanity commonly develops from chronic alcoholism, from the excessive use of the poison for a long period of time. It is three or four times as frequent as a factor in the insanity of males as of females. Usually it is not difficult to discover the cause of an alcoholic insanity, but, should alcoholic abuse be denied, an investigation of the condi-



tion of the viscera will often throw light upon the subject (cirrhosis of the liver, fatty heart, chronic gastric catarrh with heavily furred tongue, chronic nephritis, and arteriosclerosis). Corroborative evidence will generally be afforded, too, by affections of the nervous system (alcoholic polyneuritis; alcoholic epilepsy; muscular paresis here and there in the hands, face, or tongue; fibrillary tremor of the face and tongue, fine or coarse tremor of the fingers and hands; paresthesias, hyperesthesias, neuralgias; muscle volantes, titulus aurium, amblyopia, and visual hallucinations, sometimes pupillary paralysis.)

The alcoholic psychoses may be classified as follows:

Pathological drunkenness.

Delirium tremens.

Acute hallucinosis.

Korsakoff's psychosis (or polyneuritic psychosis).

Chronic alcoholism.

Alcoholic paranoia.

*Pathological drunkenness* differs from ordinary drunkenness by the manifestation of sudden strong anxious affects with blind motor explosions, or of melancholia symptoms, or of mania-like attacks. The duration is short, followed usually by deep sleep and amnesia. Pathological drunkenness is the type found among dissipated persons of all kinds, and among feeble-minded, epileptics, hysterics, neurasthenics and the like, who are generally intolerant of alcohol. The diagnosis may be aided by finding pupillary paralysis and by experimental determination of alcohol-intolerance.

*Delirium tremens* is an acute psychosis in chronic alcoholism, lasting an average of four days, with 10 to 15 per cent. of mortality, characterized by complete sleeplessness, wide-spread general tremor, disorientation as to time and place, zooscopic hallucinations, and sometimes epileptiform attacks.

*Acute alcoholic hallucinosis* is a psychosis lasting a few days or weeks, characterized by crowding hallucinations of hearing and paranoid ideas (delusions of reference, persecution, and jealousy), but without disorientation.

*Korsakoff's psychosis* is a syndrome in which are found multiple neuritis, defect of recent memories (disorder of retention), retroactive amnesia, and confabulation.

*Chronic alcoholism* presents defects of intelligence and of old and recent memory, together with moral deterioration, complete lack of insight, and occasional epileptiform attacks.

*Alcoholic paranoia* is observed in some cases of chronic alcoholism. Paranoid ideas are developed, and are more or less systematized. The most common delusion is that of jealousy or infidelity; the delusions of poisoning are also common.

**Morphin.**—Morphin is, among the alkaloïds, the most frequent cause of insanity. It is a sad commentary on the heedlessness of some medical men, but the family physician is responsible in almost every case of development of the morphin habit and its far-reaching consequences. It should be looked upon as a sin to give a dose of morphin for insomnia or for any pain (such as neuralgia, dysmenorrhea, rheuma-

tion) which is other than extremely severe and transient. The earliest symptom of morphinism is a general sensation of disquiet, manifested by incoherence of thought, difficulty of concentration of the mind, marked motor restlessness, and insomnia. The dose is gradually increased, and may reach a maximum of five or more grains.

The chief physical disorders induced by long-continued use of morphin or opium are as follows:

1. Anorexia and constipation (later, diarrhea often).
2. Cachectic anemia.
3. Cardiac weakness and intermittence, and bradycardia.
4. Muscular weakness with tremor.
5. Miosis in the early stages, mydriasis later, with sluggish reaction of the pupils.
6. Impotence. Anæsthesia in women.
7. The knee-jerks are often absent.
8. Diminished sensibility to touch and pain, and concentric limitation of the visual fields.
9. Headaches and localized shooting pains, neuralgias, and paresthesias.
10. Sensation of feeling cold.

The psychic symptoms may be summarized briefly, thus:

1. Simple elementary illusions and hallucinations, *marce volitantes*, *finitus aurium*.
2. Loss of will and æsthetic sense, irritability; moral perversion, as in alcoholic psychic degeneration, but with little failure of memory.
3. Diminished attention, incoherence of ideas, and easily fatigued intellectual powers.

A well-developed psychosis is usually the result of abstinence from morphin, and not of the chronic misuse of it. It varies in degree up to a type approaching acute mania.

**Cocain.**—Of recent years there have been numerous instances of cocain insanity, and they are doubtless growing more frequent. While with morphin it is the abstinence that is prone to induce a psychosis, with cocain, on the contrary, it is the prolonged use of the drug that develops the insanity, while abstinence gives rise to few noteworthy symptoms. The misuse of cocain leads to the evolution of an acute hallucinatory psychosis.

**Hashish (Cannabis Indica).**—We never see insanity from this cause in America, but in Egypt and India it is extremely common. In visits paid by the writer to the Cairo Insane Asylum in the winter of 1891-'92, he observed 64 cases of the 248 patients in the institution in which the insanity was due to the inhalation of hashish by smoking. The symptoms produced are indigestion, diarrhea, increased appetite, dilatation of the pupils, drooping eyelids, anemia, general debility, and delirium. The earliest mental symptom is marked and increasing timidity, sometimes amounting to *fobie des doctes*, or an agoraphobia.

**Atropin; Hyoscyamin; Hyoscín.**—These isomeric alkaloids have much the same physiological effects (mydriasis, paralysis of accommodation, dryness of the throat, depressed heart's action, dreadful illusions and hallucinations, etc.), but instances are not common of their giving



rise to psychoses. However, it is probable that the employment of one of these as a secret cure for drunkenness has been the cause of serious insanity in a considerable number of cases that have found their way from sanatoriums to asylums.<sup>1</sup>

**Metallic Poisons.**—Lead and mercury at times induce insanity, the former much more frequently than the latter. The intoxication is chronic, but the psychosis developed may be either acute or chronic. Both of these poisons produce similar psychic symptoms, such as vertigo, sleeplessness, rudimentary or marked hallucinations, confusion and incoherence, anxious depression, and often persecutory delusions. In severe cases there is dementia. In lead cases there are usually to be observed the concomitant physical symptoms, such as anemia, colic, blue line on the gums, tremor, arthralgia, palsy, and convulsions. In mercury cases we note stomatitis, tremor, and gastro-intestinal catarrh. Hysterical symptoms are not infrequently superadded upon the lead and mercury psychoses.

**Various Poisons.**—There are many other poisons which, in rare instances, produce insanity. Among these may be mentioned coal-gas, carbonic acid, stramonium, henbane, hemlock, bisulphid of carbon, etc. The writer described some years ago three cases of bisulphid of carbon insanity which ran their course under the type of acute mania going on to recovery, studied by him at the Hudson River State Hospital for the Insane.<sup>2</sup> All three were workers in a rubber factory.

**Bodily Diseases and Disorders.**—**Syphilis.**—Syphilis is one of the most important of the physical causes of insanity. It acts upon the brain indirectly through wide-spread, severe disturbance of general nutrition and through arteriosclerosis, and directly by the production of diffuse changes in the tissues of the central nervous system, or of circumscribed meningeal deposits or intracerebral granula. The degeneration of cells and fibers, the gliosis and the arteriosclerosis, are possibly due to toxins created by specific micro-organisms, and not to the direct influence of the germs themselves, which may explain why syphilitic psychoses are ordinarily late manifestations of syphilis. General paresis and cerebral syphilis are the chief phases in which the psychosis is presented. It is often difficult to obtain a history of syphilis in a patient, so that the statistics as to the frequency of syphilis as a cause of general paresis, for instance, are generally faulty. Where the history is uncertain, a careful examination may indicate the existence of syphilis (hereditary syphilis in the children, leukoderma, cicatrices, swelling of the lymph-glands, peristomal deposits and trophic perforation of the palate, nasal symptoms, etc.).

Hereditary syphilis plays a part in the etiology of the psychoses of early life,—for example, imbecility and idiocy,—though probably not so great a part as is frequently asserted, for, in a considerable experience with such conditions at the Randall's Island Asylum for Idiots, I have seen but little hereditary syphilis.

<sup>1</sup> Dr. R. D. Travis, Superintendent of the Morris Plains Asylum, N. J., has collected a number of such cases.

<sup>2</sup> *Boston Med. and Surg. Assoc.*, 1892.

**Infections.**—Typhoid fever, malaria, pneumonia, influenza, and acute articular rheumatism head the list of acute fevers which sometimes superinduce insanity. Disturbances of nutrition, high fever, and toxic changes in the blood are responsible for the symptoms developed. Perhaps the toxin-producing bacteria are the chief agents, acting by direct influence upon the cortical cells and fibers. If this be true, these cases were better classed under the head of Toxic Influences. At the height of a fever we have a febrile delirium, characterized by hallucinatory incoherence; but later on, when the fever has diminished and the organism is weakened by disease, such manifestation is termed "inamitic delirium." From either the febrile or inamitic delirium a psychosis may develop, usually assuming the type of a hallucinatory paranoia with self-depreciatory or persecutory delusions, in some cases with a tendency to agitation, in others with inclination to a stuporous condition. Manic or melancholic symptoms are rare; stupidity with a proclivity to terminal dementia, more common. Malarial psychoses sometimes exhibit a certain periodicity, corresponding to the intermittent nature of the cause. Heredity, alcoholic degeneration, etc., also play a considerable part in the etiology of this form of toxic mental disorder.

**Tuberculosis.**—The disturbances of nutrition in tuberculosis, as well as the mental depression sometimes associated with the disease, occasionally lead to the development, out of an exhaustion or inamitic delirium, of a true psychosis—a melancholia or a hallucinatory excitement. It may be said, however, that the relation of tuberculosis to insanity is much more frequently that of sequel than of prodrome, for many cases of melancholia and stuporous forms of insanity die of this disease owing to shallow respiratory functions and insufficient nutrition.

**Carcinoma.**—The progressive cachexia induced by malignant disease, as well as the direct effects of cerebral metastases, sometimes lead to psychopathic conditions resembling those of tuberculosis.

**Heart Disease and Atheromatous Arteries.**—Cardiac disease is frequently found among the insane, but its precise relation to the psychoses is obscure. Doubtless, in so far as it disturbs the circulation and interferes with cerebral nutrition, it predisposes to mental instability. On the other hand, disease of the arteries (senile, nephritic, syphilitic, alcoholic, cachectic), is a much more effective disturber of nutrition, and at the same time gives rise to serious focal lesions, such as miliary aneurysms, thrombosis, and hemorrhage, which may be etiologically associated with various psychopathoses.

**Nephritis.**—The nephritic psychoses assume usually the type of a hallucinatory paranoia, and therein resemble other toxic insanities. It is probable that toxic changes in the blood are here of more importance than the changes in the vascular walls, though these, too, have their significance.

**Gastro-intestinal Disorders.**—These sometimes induce hypochondriacal melancholia, and predispose to psychoses of various kinds by disturbing nutrition; but their frequent relation to insanity is generally a consequence rather than a cause.

**Diseases of the Genital Organs.**—There are serious disorders of



the female genital organs which occasionally play a rôle in the causation of insanity, but their importance as factors has been grossly exaggerated, and much harm and little good have followed operative interference for the relief of the insanity. Probably the cessation of menstruation (usual in acute insanities) has been misinterpreted as significant of genital disease, and thus given rise to a grave error.

I would not be understood as decrying operative or other treatment altogether, if such be indicated; but let no one be deceived into expecting benefit from the procedures, except in rare instances.

Having briefly examined most of the general physical disorders which are concerned in the causation of insanity, we will now turn our attention to certain functional diseases of the nervous system which, by reason of their localization in the cerebral cortex, are prone to assume a very important part in psychopathology. These are epilepsy, hysteria, and chorea.

**Epilepsy.**—Epilepsy is almost as common a disease as insanity itself. Asylum physicians, whose experience with epilepsy is limited to cases associated with mental disorder, tend to overestimate the frequency of insanity among epileptics. Thus, it is often stated by them that psychic degeneration is manifested in sixty to eighty per cent. of all epileptics. But the fact is that probably not more than ten to fifteen per cent. of epileptics develop insanity; at the same time the proportion is so large as to show a close relation between this functional cortical malady and mental disorders. When progressive epileptic degeneration occurs, it manifests itself by the following symptoms:

1. Slowness of ideation and articulation.
2. Abnormal irritability of temper.
3. Hypochondriacal depression.
4. Paranoid outbreaks of various kinds.
5. Dementia.

**Hysteria.**—Hysteria is also a functional neurosis of the cortex, often closely associated with diverse psychoses. There is a species of hysterical psychic degeneration, and the neurosis frequently gives a special odor to different forms of insanity. The symptoms noted (aside from the peculiar sensory and motor manifestations familiar to us in simple hysteria) on the mental side are as follows:

1. Lack of logical coherence and sequence of thought, but with perfect intelligence. Defects of memory, with rudimentary persecutory and erotic delusions, are encountered frequently.
2. Extreme uncontrolled and morbidly changeable emotions. Profound egotism.
3. Frequent illusions; occasional hallucinations.
4. Conduct and speech are based upon emotional impulsiveness, uncontrolled by ethical considerations of any kind.

**Organic Nervous Diseases.**—The psychic disorders induced by organic processes in the brain, such as meningitis, tumor, softening, hemorrhage, and the like, are characterized either by symptoms of retardation of functions or by symptoms of irritation, and are due either to pressure or to the indirect influence of the lesion upon the circula-

tion of nerve-centres and tracts. Emotional irritability, hallucinations of the various senses, defects of intelligence reaching to imbecility or idiosy, stuporous conditions—these are common mental manifestations of such processes. Since single localized lesions are apt to produce slight mental changes, any marked intellectual defect or multifarious psychic symptoms may be looked upon as suggestive of wide-spread, perhaps multiple, lesions, such as multiple sclerosis, multiple tumors, syphilis, etc. Sometimes true insanities develop in these cases, particularly when there is hereditary instability of nervous organization.

**Traumatic Insanity.**—When we speak of traumatic insanity we exclude everything except trauma to the head. There are cases of mental disease resulting from operations such as ovariotomy and other laparotomies, from merely taking an anæsthetic for an operation, and from railroad accidents and the like where there are injuries to other parts of the body than the head. There are also many cases of tumor of the brain depending upon trauma to the head, in connection with which mental symptoms, even insanity, may develop.

An injury to the head may be so serious as to produce local symptoms such as extensive hemorrhage, laceration of the brain, paralysis, aphasia, coma, and the like, and with such conditions there may also develop mental symptoms, including insanity. This type of insanity corresponds to what is known as post-apoplectic insanity and is also excluded from the category proper of traumatic insanity.

Injury to the head not infrequently induces epilepsy, and epileptic attacks if frequent and severe induce an epileptic dementia, but these cases do not fall into the classification of traumatic insanity.

Trauma to the head like trauma elsewhere may bring about hysterical symptoms only, such as we are familiar with in the traumatic neuritis.

While injuries to the head are very common, insanity as a result of it is very rare. For instance, in the Franco-Prussian War out of 1985 soldiers wounded in the head only 13 became insane. Stolper collected 981 injuries to the head, and of these, 138 had a very serious concussion. Yet only 12 cases of insanity resulted from these injuries.

In a consideration of traumatic insanity we must make a thorough investigation into antecedent conditions, such as hereditary predisposition, previous attacks of mental disorder, an alcoholic history, and the presence of syphilis. The trauma may simply be a determining factor in an attack of mental disease, an outbreak of delirium tremens, or the beginning of paresis.

Traumatic insanity then has a very restricted place in the classification of insanity.

Suppose we limit ourselves to what we may call a concussion of the brain from a blow, shot, or fall, without fracture or laceration, but believing, of course, that there undoubtedly is some structural disarrangement of cells and fibers, punctate hemorrhages, or even possibly extravasations of blood too small for focal symptoms. The severity of the trauma seems to have very little relation to the ultimate seriousness of results, an extensive injury often being quickly recovered from, a comparatively



mild trauma as often inducing disastrous consequences. The part of the cranium struck is sometimes indicated by the syndrome of symptoms. For instance, a blow to the occiput may induce a cerebellar syndrome. A blow on the frontal region may give rise to symptoms wholly mental. So that at times the manifestations of disorder are due to injury of parts directly beneath the point of injury. But more often the symptoms presented are due to indirect rather than direct results of the trauma—diffuse or general concussion, congestion or other injury at a point opposite the site of the blow, or at points in any part of the encephalon where the tissues have a different specific gravity.

The symptoms of trauma to the head may be temporary or long-continued, moderate or severe.

The concussion may give rise to complete loss of consciousness or the condition of being confused or dazed. In a mild trauma the patient may merely show pallor, vertigo, confusion, and may stagger or fall. After a moderate injury there may be complete recovery in a short time or there may remain headaches and easy fatigue from exertion. A severe concussion causes signs of shock, such as great pallor, weak and rapid pulse, cold and moist hands and feet, with a varying degree of unconsciousness, from complete coma to a light stupor. If there has been no marked organic injury to the brain itself or intracranial hemorrhage there will be no sign of paralysis, though there are sometimes convulsions. The pupils are usually dilated, though occasionally contracted, and the pupil may be dilated on one side (the same side) in unilateral hemorrhage. It is quite possible that even in the more moderate cases, while there is no gross lesion of the brain or meninges we may have punctate hemorrhages. In moderate trauma to the head consciousness returns in a day or two as a rule, but headache and vertigo persist, and it is doubtless in the cases where we have to do with punctate hemorrhages or other minute structural changes in the brain that mental symptoms arise. Mental confusion may last a long time with amnesia, and it is not infrequent to observe a delirium. The patient may show depression, irritability, and retarded mental activity. When delirium is present it is possibly due to a certain amount of inflammatory reaction, a local meningitis, or circumscribed encephalitis. We may have a permanent mental weakness as a result, a traumatic dementia.

Even where an individual seems to have recovered fully from the effects of a concussion of the brain, or a *commotio cerebri*, as it used to be called, he may be more vulnerable to the various toxins of disordered chemistry or of micro-organisms, and is apparently always intolerant of alcohol.

In some cases immediately after a partial recovery from the coma we have more than a simple delirium, viz., an acute excitement, with motor agitation, extreme dissociation, from which the patient may slowly recover in a few weeks or months. In certain patients hallucinations are so marked that they are called cases of traumatic hallucinosis, and in these not infrequently states of stupor or even katatonic syndromes supervene.

Another group of cases is characterized rather by a more or less apa-

thetic, dull and stuporous state, lasting a long time, without delirium or hallucinations, with marked defects of memory, great retardation of thought, and a kind of approach to a condition of double consciousness.

All these cases just referred to relate rather to immediate syndromes resulting upon trauma. There are late degenerative effects of injury to the head, developing long after subsidence of the initial somatic symptoms. These symptoms may be neurasthenic or psychasthenic, or there may be marked change of character with great irritability, emotional explosiveness, headache, cephalic paresthesia, and vertigo. The Friedmann syndrome is a common one. This is a traumatic vasomotor neurosis characterized by headaches, vertiginous attacks, feeling of fullness in the head, sudden pallor, and marked intolerance of alcohol.

The amnesias that follow concussion of the brain are various, sometimes single amnesia, sometimes retrograde or anterograde, or both.

**Exhaustion.**—Stresses of various kinds, mental or physical, especially in conjunction with the impairment of the nutrition of the central nervous system, induce an exhaustion upon the basis of which a psychosis may develop. The mental strain may be from overwork, overstudy, insomnia, and the like; the physical, from masturbation, sexual excess, hardships. The nutritive impairment is the result of some blood-change or deficiency, such as constitutional anemia, a cachectic state, etc. The physical symptoms of such exhaustion are: slowing of the thought processes, difficulty of recollection, want of ability to concentrate the attention, rapid fatigue on mental exertion, emotional irritability with an undertone of depression, leading often to fully developed insanities, which are designated as asthenic psychoses. The common features of such psychoses are retardation and incoherence of the mental processes, unmodified even in the quality of the hallucinations and delusions. But almost any form of insanity may be evolved from this asthenic state of the nervous system, such as mania, neurasthenic, stuporous states, and various paranoid conditions.

As Ziehen points out, it is also interesting to observe how any exhausting psychosis may in itself induce this asthenic condition with the characteristic features of an asthenic psychosis, as a result of which we have a secondary type of mental disorder developed upon the basis of the original insanity.

**Physiological Factors.**—Puberty, the puerperal state, the climacteric, and senility are indirect strains to which the organism is subject, by reason of the more or less profound physiological commotions they arouse in the nervous system—commotions which may well disturb the normal adjustment and equilibria of the thousands of delicate processes going on in the brain, and thus enormously increase its vulnerability to the direct factors which beget insanity.

The curve of psychic morbidity reaches its highest points, corresponding to maximal aggregations of etiological factors in both sexes, at puberty, middle age, puerperal periods, the climacteric, and senility.

**Puberty.**—From the thirteenth to the twentieth year there are remarkable changes, physical and mental, in the growing individual. These are more noteworthy in the female than in the male sex, for the



time is shorter for the change from girlhood to womanhood than from boyhood to manhood. The evolution of the sexual characters and the development of the powers of reproduction induce a stream of innumerable new stimuli from the genital organs to the brain, accompanied by wholly new organic sensations, new associations, and new and powerful emotions. The evolution is rapid, and, as is the case with all rapid development, more or less unstable.

The boy grows fast in body, takes on the aspect of manhood, with a stronger and more rugged frame, a changing voice, a budding beard. His mind is filled with new sensations, emotional, sentimental, amatory, and with clanging, fantastic, illusory dreams and imaginings. Even in the normal youth this nascent state, this struggle of the emotions, thoughts, instincts, impulses for new associations and new combinations, may be greatly aggravated in many cases by masturbation, in others by nutritive disorders. If this be so with the normal individual, how much greater must be the stress of puberty to the individual with a constitution vitiated by hereditary taint!

The girl leaps more quickly into her place in life. The physical changes are more rapid in her, and at the same time more varied and noteworthy. It is a time of tumultuous activity of mind and body in an organism which has not the numerous outlets for surplus energy possessed by the other sex.

The psychoses of puberty are various in their expression. They may manifest themselves as a mania, a melancholia, a paranoia, or as an insanity with peculiar color, to which the name hebephrenic modification has been given; so that we speak of a hebephrenic mania, a hebephrenic melancholia, etc.

By the designation hebephrenic is understood the following syndrome: Extraordinarily rapid and paradoxical changes (depressed ideas in the midst of hysterics, gaiety, jocularity in the deepest depression), with paradoxical facial expression and gaminism; exalted motor activity (laughing, dancing, grimacing, exhorting after the manner of an orator, often with incomprehensible words and sentences); conduct and action without apparent object, but often with the semblance of desiring to attract attention.

**Puerperal State.**—Pregnancy, parturition, and lactation diminish the vitality of woman, debilitate and weaken her entire organism, induce a species of physiological exhaustion in her nervous system, and, in short, bring to bear a strain upon her which is, even under normal conditions, attended by emotional irritability, depression, morbid yearnings, etc. It is not strange, therefore, that the puerperal period in women with unstable nervous systems should often be an exciting factor in the development of psychoses of various kinds. In about ten per cent. of insane women the insanity has its origin at the epoch of reproduction. The majority of these cases are parturitional (seven and a half per cent.), while about a fourth are lactational and a tenth pregnancy cases. It is perhaps true that there are many cases of parturitional psychoses in which the insanity is not so much due to the stress of labor as to possible auto-intoxications from poisonous substances

absorbed during the catabolic changes incident to subinvolution of the enlarged uterus. As important factors, too, we must include loss of blood, parametritis, sepsis, mastitis, etc.

There are divers forms of insanity consequent to the puerperal state, such as acute hallucinatory paranoia, melancholia, stuporous insanity, mania, and neurasthenic insanity.

**Menopause.**—The climacteric, between the ages of forty and fifty, is another epoch of change in woman, a period of involution in its way analogous to the evolution at the age of puberty. There is a physiological commotion in the nervous system at the time of the cessation of ovulation and menstruation, a disequilibrium associated, even in normal individuals, with numerous neurotic manifestations, and, in such as have unstable organizations, attended with peril to the mental integrity. Melancholia, simple and hallucinatory paranoia of chronic character, and circular insanity are the forms of psychosis incident to the menopause. About four per cent. of the cases of insanity in women are due to the climacteric.

**Senility.**—The involution of all the tissues of the body characteristic of especially the seventh decal of life forms also a frequent basis for insanity which depends, in the main, upon the loss of functional activity in the cerebral cortex. Such loss is notable even in normal individuals. The latest acquisitions of the mind are the least stable; hence the conspicuous loss of memory for events of recent occurrence and the tendency to live in the past. The scope of interests, sympathies, and ideas narrows itself down to the individual's immediate physical comfort and needs. While the physiological involution of senility belongs to the seventh decade in many instances it begins long before this, owing to general debility, endarteritis, etc. Marked changes in the brain of such nature must, therefore, often superinduce veritable psychoses in individuals predisposed to mental disorder by heredity or by antecedent physical or psychic stresses.

Senile insanities manifest themselves in many forms,—melancholia, mania, incoherent paranoia, hallucinatory paranoia, dementia,—but, of course, modified from the common types by the weakening of the cerebral functions previously described. Vertiginous seizures, slight parestheses, dreadful hallucinations, and primary anxious conditions are often observed in all of these forms.

The hallucinations appear in senile forms in psychoses which ordinarily run their course without them, and the anxious states in ordinarily non-affective insanities.

**Moral Causes.**—About twenty-four per cent. of all cases of insanity are ascribed to moral causes, among which are classed domestic troubles, grief over death of friends, business reverses, anger, religious excitement, love affairs, fright, and nervous shock. The percentage is greater in women than in men. It is doubtful if any emotion alone can overcome the stability of the normal nervous system; hence it is in the fragile, nervous constitutions of individuals tainted by heredity that extreme emotions are wont to exert their malign influence.

The uncertain equilibrium of the highest nerve-centers in these cases



is all too readily overcome by the tumultuous wave of an intense emotional impression. Possibly, the results depend upon disturbance of the vascular innervation. Ordinarily, the greater and more sudden the emotion, the greater the liability of the badly poised brain to succumb; but, like the drops that wear away the stone, an emotion of less intensity may, by long continuance, produce equally disastrous consequences. Some acute psychoses may be suddenly developed by fright, or a transitory emotional insanity for a few hours or for a few days in duration. Among the symptoms are mutism, or incoherence, confusion, isolated hallucinations, delusions, with impulses to violence and aimless wandering, followed later by complete, or nearly complete, amnesia.

The more slowly working affects, like sorrow and worry, often aid in the evolution of melancholia, paralytic dementia, or acute or chronic paranoia.

Herbert Spencer long ago showed that our life is a series of constant adjustments of internal relations to external relations, and it is only of late that we have begun to apply his lucid explication of this process of the normal mind to our study of what goes on in the abnormal mind. This has led to a new valuation of the moral cause, by which we always meant mental cause, and for which we now employ the adjective *psychogenic*. We have come to believe that insanity is more often of psychogenic origin than we formerly supposed. This may have its rise in some psychic trauma, some sudden shock or profound emotion, or it may start from some minor maladjustment of "the internal relations to the external relations," some small disorder of the individual's reactions to the realities about him, leading to a whole series of wrong adaptations that finally result in actual mental disorder. The result of this sound view is a more thorough study nowadays of the patient as an individual, rather than as an ordinary type of insanity, and, consequently, a better system of therapy.

**Imitation.**—The so-called psychic infection never influences normal individuals who are brought into contact with the insane. Physicians, attendants, and others who have to do with the insane are never affected, except when morbid heredity and mental and physical overwork combine to prepare the soil for the development of a psychosis. The writer recalls but one instance of an attendant being mentally unbalanced during her service. She was neurotic by constitution and cut her throat a few days after one of her patients had committed suicide in the same manner. But there are not infrequent instances of communicated insanity among members of a family.

The simultaneous development of insanity in two or more persons associated together, or the imposition of delusions gradually arising in the mind of one upon the impressionable intellect of a second, third, or of many persons, has been described under the names *Folie à Deux*, *Folie à Plusieurs*, *Reciprocal Insanity*, *Folie Imposée*, etc. There are several factors which govern the evolution of such insanities. In both forms a degenerative soil is usually required for the proper germination and growth of morbid ideas. In the simultaneous variety there must be, in addition to predisposition, that similarity of intellectual substrata which

we find particularly in persons who are blood-relations or who are intimately joined together by mutual like and dislike; hence it is that brothers or sisters most frequently manifest simultaneous insanity. Take two healthy children of one family and bring them up far apart, yet there will be innumerable physical resemblances between them, and many peculiarities in their character and conduct which prove them to be consanguineous; if a hereditary instability of nerve-cells had been implanted in them, there would be a tendency to a similar form of dissolution, even if they remained apart. How much greater would this be in two persons so intimately associated as sisters, for instance. In children the study of unconscious imitation is one of great interest. Who has not observed the identity of intonation of phrases, of posture, of laughter, of many facial expressions, of certain habits, in children either related or brought up together? Such unconscious imitation, as is well known, may lead in children to the contraction of certain nervous and even mental diseases. The contagious quality of emotions is well established. An explosion of laughter will call up smiles on even melancholy faces in a crowd. A pathetic scene on a stage will bring tears and depress the oral angles in a large audience. The unconscious imitation of gestures, such as bowing, often seen in adults, is in a milder degree such mimicry of motion as is observed in dancing mania.

Another element in the imposition of insanity by one upon another is the quality of the morbid mind-product. If a delusion, it must have an air of probability to the person receiving it, and must be gradually developed and imposed. It is because suspicion is inherent in the nature of most persons, because suspicion can wear so much probability of truth, that persecutory delusions are by far the most frequently adopted by others. Credulity is an important factor in the imposition of insane delusions upon others. It was the ready credulity of large numbers of persons, especially as regards religious subjects, that in the past led hundreds of thousands of people to adopt with faith the delusions of paranoiacs like John of Leyden, John Thom of Canterbury, Jean of Arc, Richard Brothers, Joanna Southcott, John Brown, and many others, and actually to sacrifice their lives upon the altar of their beliefs. Though these delusions emanate from an insane person, their acceptance by others does not, of course, necessarily imply insanity in the latter, for delusions of this character have their support in the superstitions of many and in ignorance concerning supernatural matters. A persecutory delusion might be imposed by an insane person upon an intimate associate, and yet the latter need not, of necessity, be insane; but when the exposed individual adopts the delusions, regulates his conduct upon them, allows them to become rooted in his mind, even begins to share the hallucinations of his friend, there is, of course, actual aberration of mind present. Several cases of *folie à deux* have come under my observation. One case was that of two sisters, aged about fifty to fifty-five, Irish, washerwomen, who, living alone in a tumble-down slum, were often tormented by boys throwing stones at the house at night, and otherwise teasing them. They finally developed persecutory delusions with hallucinations, and both were very much



alike. They became so violent in their demonstrations that ere long both were taken to the asylum, where I took charge of them. They were separated, the result being that one became rapidly demented and the other became a quiet worker, with fixed persecutory ideas and auditory hallucinations.

Another pair of sisters, colored, between forty and fifty years of age, were similarly affected. For ten years one sister had been a paranoiac, with delusions of persecution by means of electricity, which was at all times, night and day, hurled through her body by a vast organization of conspirators. She had hallucinations of hearing. The sisters had not lived together until within six months of my seeing them, the same sister having recently become a widow. The same sister gradually adopted the delusions of the insane one, and probably the hallucinations. She believed her sister to be persecuted by an organized band of conspirators with electrical appliances.

A third case was that of a husband and wife, who both became typical cases of melancholia, with, of course, similar delusions, one shortly after the other. Such a case as this might be called a coincidence, and not an imposed insanity. Probably grief over the insanity of the husband was one factor in developing that of the wife, but unconscious emotional imitation between two persons united by special bonds of sympathy was undoubtedly another element.

A fourth example I detailed fully some years ago in the "*Alienist and Neurologist*" ("Paranoia in Two Sisters," January, 1890).

C. K. and H. K. were respectively thirty-six and sixty-two years of age, teachers of music and singers by occupation, of German parentage, and had both been insane for some ten years. Their mother was a case of paranoia, with fixed delusions of an exalted religious nature. She believed herself to be the mother of God. She was never in an asylum, but lived at home until her death. While insane she gave birth to the younger of the two sisters, C. K.

One of them wrote for me an autobiographical sketch, and the other some twenty-five letters, upon which the following facts in their identical clinical history are based:

The instigators of the conspiracy against them are chiefly their uncle, brother-in-law, and sister-in-law, and a brother has also been inveigled into it. By them are employed numerous detectives, expert chemists, and handicraftsmen, and, as they have privately hinted to me, also many lawyers. Openings are made in their rooms in spite of all they can do for the insufflation of noxious gases, smoke, camphorums, chloral, and chloroform vapors; and by some unseen agency substances are thrown at them which produce painful cutaneous eruptions. Their food and water and heating apparatus are tampered with for the introduction of poisons or to produce serious illness. They hear the mechanics at work upon the floors, walls, ceilings, and the voices of the detectives (hallucinations of hearing). Their food has a peculiar taste (hallucinations of taste). Most prominent of all are the singular odors of the room, of fruit and flowers sent them, of the water (hallucinations of smell). Sometimes they are black in the morning when

they look in the mirror (illusion of sight). They are subject to remarkable, generally painful, sensations in their bodies (hallucinations and illusions of cutaneous sensibility).

They hint of imaginary property in Germany, out of which they are being defrauded by relatives. For ten or twelve years they have been driven from one place to another in Brooklyn and New York by their pursuers. As yet they have sought only escape and protection from persecution; they have very rarely manifested anger by pounding the floor when hearing the mechanics at work or by complaint to the landladies, and have not been brought to bay to a condition in which they might turn upon the actual instigators of the conspiracy and do them bodily harm. They have been on the point of a visit to police headquarters to make declaration against their enemies.

From what I can learn of their history in youth the two girls differed from others of their age in a slight degree, some trifling eccentricities and some overpowering self-consciousness constituting this difference. They have always been closely united—living together, sleeping together, having the same affinities, talents, pleasures, and pursuits. The development of suspicious and delusions of persecution had been so gradual that it did not become evident to others that they were actually insane until a comparatively recent period. When I first saw them in my office, they came heavily veiled, and, upon removing their veils, their faces were patched all over with small square pieces of cloth, covering sores. These were only an ordinary acne, made much worse by picking, by wearing wet cloths on their faces all night for the purpose of preventing poisonous vapors from entering their lungs, and by the removal of the strongly adhering pieces of linen from the bleeding surfaces. They healed up rapidly when I had prevailed upon them to make use of *ung. zinci ox.* freely. The face of the younger is particularly characteristic of a degenerate type, one of its features being a disagreeable prognathism.

Some of the skull diameters were pathological in character. One of the sisters died in convulsions from unknown cause, which the other sister still attributes to poison. The living sister still moves about from one part of the city to another, cherishing the paranoid delusions, but supporting herself in part by teaching music.



## CHAPTER III.

## GENERAL SYMPTOMATOLOGY OF INSANITY.

EVERY psychic phenomenon is accompanied by a material process in the cortex of the brain. There is no insanity without disease of the cortex. The material disorder of the cortex is diffuse and partly organic, but mostly functional in character. We term it functional, for thus far our pathologico-anatomical and clinical studies have failed to reveal any definite material basis for the majority of psychoses.

The progress made of late years in the study of physiological psychology has illuminated many obscure features of morbid psychology, and has put us in a position to better examine and classify the symptoms of insanity.

There are material processes in the central nervous system unaccompanied by any parallel psychic process. The reflexes and automatic acts are examples. In these phenomena we observe a stimulus, a sensation, a movement. Movement paralleled by a psychic process becomes action. We sometimes speak of conscious voluntary action. Action differs from simple movement in being accompanied by intercurrent images—memory-pictures of former stimuli. A peripheral stimulus excites a cortical center, and is not carried at once to the motor region, but travels first by association fibers to the area in which are stored up residues of former similar stimulations, and later to the motor region. These residues of memory-pictures or ideas may be complex, constitute a series, have many associations, and hence we designate them as an idea-association. Action, therefore, consists of the series: stimulus, sensation, idea-association, movement. The various ideas thus excited tend to different motor expressions, so that the resulting movement or action will depend directly upon the strength of ideas. The stronger ones conquer. Ziehen, whose clear explication of the mental problems of psychiatry the writer closely follows,<sup>1</sup> has well described idea-association as the play or battle of motives. He gives the following example of the physical and psychic processes just described:

I see a rose in a strange garden (stimulus and sensation). A long series of ideas is aroused by the stimulus and the visual sensation of the flower (idea-association). For instance, the memory of the rose's fragrance comes to mind, then I think how well it would look in my room, that it is the property of mother, that plucking it would be punishable, and so on. Only after the whole series of presentations has passed before the mind does action follow, and whether I pluck the flower or go my way without it will depend upon the strength and intensity of the conquering idea.

Every psychic process must be regarded upon the basis of such a

scheme, and as accompanied by its material parallel (progress from the sensory cells to the idea-cells, and from these to motor-cells by means of association-fibers).

Sometimes the idea of movement (memories of former sensations of movement) comes before the movement in the series just described, but generally the movement is perceived after it has taken place by means of the sensation of the movement.

There are really but two psychological elements—*viz.*, sensation and idea. The only process connected with these elements is the idea-association. Their product is action. The so-called mental powers of old-time psychology do not exist. The assumption of a special power of will dominating the idea-association and voluntarily determining this or that movement is particularly superfluous and misleading. The assumption of a special power of apperception which turns its "attention" voluntarily upon this or that idea or sensation to determine the course of the idea-association is equally superfluous.

The presentations or ideas rather follow one another according to laws without intervention of any especial voluntary power of the mind, and the final movement or action is the necessary result of association of these presentations or ideas. Finally, there exists no particular faculty of feeling, for exact investigation demonstrates that our feelings of what is agreeable and what is distasteful, of pleasure and pain, appear never in an isolated state, but always combined with sensation and idea as attributes or properties.<sup>1</sup>

Following Ziehen in these particulars, we shall study pathological psychological processes on the basis of the scheme just described, and in each case investigate, first, disorders of sensation; then, disorders of the memory-pictures, presentations, or ideas; then, again, disturbances of the idea-association; and, finally, the influence of these disturbances upon the actions or conduct of the patient.

#### DISORDERS OF SENSATION.

Sensation is the first element in the psychic process. It is determined by some external stimulus affecting any sensory nerve. Every sensation has four important attributes—*viz.*, quality, intensity, tone (the accompanying feeling of pleasure or pain), and space-projection. We are not especially concerned with the last in morbid psychology.

**Qualitative Disorders of Sensation.**—The two important classes of qualitative disorders of sensation are *hallucinations* (in which we have sensation without external stimulus) and *illusions* (in which we have the external stimulus, but a transformed or perverted sensation). An external stimulus to a peripheral nerve is carried to the cortex, where it acts as a secondary stimulus in exciting sensation.

**Hallucinations.**—A hallucination is a sensory impression without external stimulus. It is often also defined as a perception without an

<sup>1</sup> "Leitfaden der physiologischen Psychologie," von Th. Ziehen, Jena, 1903.



object. The patient hears voices where all is silent, sees forms and figures in empty space.

Hallucinations of sight are very common, and vary from the simplest sparks, lights, shimmers, flames, spots, threads, clouds, and shadows to the most complicated groups of persons and landscapes with perfect details. Sometimes they are colorless, like silhouettes; sometimes radiant and fantastic with color. Sometimes they are flat, like pictures; sometimes plastic. Ordinarily, the forms and objects observed are of natural size, but occasionally they are gigantic or diminutive. They may appear close at hand or far away. They may be quiet or full of movement, like the zooscopic hallucinations of alcoholism. Rarely, real objects are doubled or multiplied (hallucinatory diplopia and polyopia). Real objects are sometimes concealed by the hallucinations, sometimes merely diaphanously veiled. Hallucinations may fill the whole field of vision or appear in homonymous half-fields, as in the hemipic hallucinations described by the writer in cases of insanity and of homonymous hemianopia.

Hallucinations of hearing are also extremely frequent, and vary from simple sounds, tinnitus aurium, rushing, roaring, whispering, tinkling, to complicated music and words and sentences. These last may be in natural tone or deep-voiced, whispered or loud; may be the voice of one or many persons; may be pronounced in various languages; may be single words or long sentences; may seem near at hand or far removed; and may be heard in one ear, though usually in both. Not infrequently the voice seems to the patient so near that it appears to be in his head or body.

Hallucinations of common cutaneous sensibility may appear anywhere in the skin or in mucous membranes in the form of electric shocks, pricking, tingling, itches, caresses, sensations of heat or cold, indignities to the sexual organs (feeling of cohabitation), etc.

Hallucinations of smell are very common. The patients perceive odors of chloroform, sulphur, noxious gases, smoke, filth, or, on the other hand, the smell of perfumes and flowers.

Hallucinations of taste are so generally combined with those of smell, because of the close physiological relation of the two senses, that true hallucinations of the primary elements of taste (salt, sweet, bitter, and sour) are uncommonly rare. A hallucination of a bitter taste is the most frequent. On the other hand, the combined hallucination of taste and smell (as of blood, filth, etc.) is rather common.

Hallucinations of organic sensation are not rare. The patients complain of peculiar or extraordinary feelings in various organs, such as malposition, growing, cutting, pain, etc.

Hallucinations of active or passive movement of the body or its parts depend probably chiefly upon disorders of joint sensibility. The patients feel themselves lifted in the air, floating, the limbs moved actively, the head turned to one side; or, the sensation of movement of the muscles required for speech may give rise to the hallucination of having spoken a word or sentence.

Various hallucinations are often associated in such a manner as to render the hallucinated objects still more natural and deceptive, though more frequently they are not thus commingled. Thus, visionary figures may speak or be dumb, and the fancied voices may come from visually projected or from unseen persons. Sometimes vision, hearing, and cutaneous sensation may be combined to give reality to the object. Combinations of others are also met with, and, indeed, these mixed hallucinations are common and multifarious.

As regards the development of hallucinations, some are doubtless peripheral, but the majority are central in their origin. Disorders of the eye, ear, nasal cavity, mouth, mucous membranes, skin, and viscera may give rise to hallucinations, though they are more commonly the cause of illusions. Hallucinations are never new creations, but are made up of memory-pictures stored up in the cortex; these may, however, make their appearance in new combinations. The congenitally blind never have visual hallucinations; the congenitally deaf never auditory hallucinations, though they are noted in acquired blindness and deafness.

Hallucinations are usually of two kinds—those which have to do with the ideas presented in the mind at the time of their manifestations, and those which are concerned with latent memory-pictures. The former are more common, but both may be observed in the same patient. The first kind are those which the patient describes as visions which picture his very ideas, and voices which read off his thoughts as fast as they can come into his mind—indeed, often apparently before he thinks them. The second class of hallucinations often astounds the patient by association with things long past and quite forgotten.

We are taught by physiological psychology that a stimulus to the eye arouses a sensation in the occipital lobe, to the ear a sensation in the temporal lobe, and so on, the sensation further exciting an image which remains as a memory-impression. All normal sensations, then, depend upon the series stimulus, sensation, memory-picture, or idea. Now, hallucinations are always cortical as regards localization, and depend upon a reversal of the normal course just described, and without the stimulus. The memory-image is excited and then excites the sensation. A certain irritability of these centers will be induced, undoubtedly, by morbid processes in the peripheral nerves or their terminations, such as entoptic or entonic processes, which will render them all the more excitable, since external stimulus is not then altogether wanting. Finding such,—and we should always investigate carefully for a peripheral physical basis,—the dividing-line between hallucinations and illusions becomes less distinct. Naturally, the normal mind recognizes the real nature of various volutaries, tinnitus aurium, neuralgic pains, etc., and it is only the abnormal mind which employs them as material for illusions and hallucinations.

In the examination of a patient we must determine the presence of hallucinations and the effect of their presence on idea-association. One



must not mistake actual occurrences described by the patient, nor the events of dreams confused by him with events of waking, nor ordinary illusions for hallucinations. There is danger, too, of overlooking their presence. Patients conceal them, conscious that the hallucinations are morbid, or knowing that they will be looked upon as such, but will often write about them or tell of them to other patients if opportunity be given. Very often the physician is enabled to recognize their existence from the expression and conduct of the patient.

As regards the influence of hallucinations upon the course of idea-association, the most important question is whether they are regarded by the patient as real sensations or not. He treats them as actual phenomena, as if they were normal sensations, or he distinguishes them from his ordinary sensations as peculiar, novel, and possibly inspired by supernatural agencies; or he is really conscious of their morbidity, but may believe them to be induced by enemies by means of poison. If the hallucinations are faint and transitory, the patient may not be much influenced by them; if they are marked and persistent for a long period, he ultimately loses his critical faculty and comes to believe in their reality. Such being the case, his thought and conduct are bound to be influenced by them, and more powerfully influenced than by normal sensations, or by any reasonable consideration or argument. Hallucinations either inhibit (hallucinatory stupor) or retard (hallucinatory confusion) the idea-association; or they induce direct intrinsic delusions (as when a voice cries "Thou art God," and the patient immediately believes himself to be God). The actions and conduct of a patient are very much influenced, and in multifarious ways, by hallucinations. He has the expression of listening, or stares apparently at nothing. He closes his ears, covers his eyes or head, closes up cracks and openings, or listens at the window or keyhole. He refuses or spits out his food. He holds his nose, or suddenly closes the window to prevent the entrance of noxious gases. He turns his head, runs, shouts, lifts his arm quickly, or takes peculiar attitudes, acting upon a hallucination of muscular sense (imperative movements, imperative speech, imperative attitudes). The imperative attitudes may be very persistent and long-continued, and are then called catatonic. Hallucinations often lead to imperative acts which may be of a violent nature. If hallucinations are innumerable, very changeable, and intense, the patient is affected by so-called hallucinatory agitation.

Hallucinations are so extremely rare under normal conditions that they are to be considered as almost always pathological. Illusions are rather common in the normal mind. True hallucinations may occur in apparently normal individuals, but examination will show that such persons are neurotic by heredity, and that some stress of mind or body has induced this psychopathic phenomenon. This is particularly true in childhood.

Outside of the psychoses, hallucinations are met with in toxic states, fevers, exotoxic conditions, sun-stroke, and some of the neuroses (epilepsy, chorea, hysteria). A hallucination of any sense may be the aura of an epileptic attack; sometimes, when visual, it may be hemispheric.

Hallucinations are the chief symptom of one form of paranoia. Other psychoses, such as mania and melancholia, manifest them only exceptionally; while still others, like senile and parietic dementia, present hallucinations, it is true, but not in such prominence as to make them a characteristic symptom. Visual hallucinations are more common in acute than in chronic psychoses, and they are seldom independent of hallucinations of feeling and hearing. Auditory hallucinations, on the other hand, are more characteristic of chronic types of mental disorder, and are often observed alone.

The close union of the auditory center with the motor speech center gives a peculiar interest to hallucinations of hearing. From infancy man is trained to think to a great extent in word-images or speech-images, and thinking is, therefore, nearly always associated with some stimulation of the speech-muscle centers in the brain. Therefore, hallucinatory irritation in the auditory area of the brain causes synchronous irradiation to the motor speech center, and words and sentences are heard by the hallucinant as if projected into the external world, or into some part of the patient's body (head, throat, chest, stomach, or even extremities). The stimulation of the speech muscles, however feeble, may be sufficiently strong to induce recurrent sensations of movement in these, which leads the patient to imagine that his thoughts are being read off internally by the voice, and sometimes repeated apparently before the thought has fully developed in his brain.

**Illusions.**—An illusion is a false perception. There is a stimulus but a perverted sensation, a wrong interpretation. The sensation corresponds only in part to the stimulus. A patient hears the rain falling, but perceives it as music; he sees the bedpost, but imagines it a ghost.

Visual illusions exhibit a transformation of form, or color, or both. This is often favored by indistinctness of outline, as when it is half dark or there is a shimmering, flickering light. But often clear outlines are transformed. The patient may see the familiar faces about him changed into those of strangers, transformed by grimaces, or deathly pale. A sharp distinction between illusory transformation and actual hallucination is often difficult to draw. It is peculiar to illusions that they not infrequently present objects as distorted and diminished or increased in size. This is especially true among epileptics. When this is noted with all objects, it often depends upon catoptric disorders. Thus, metamorphopsia may arise from astigmatism and retinal disease, micropsia from spasm of accommodation, and macropsia from spasm of accommodation. When this is not the cause, perverted association of the sensation, with disordered muscular sense, may play a rôle. Sometimes, though rarely, the illusion may consist of a perversion of color analogous, for instance, to the yellow appearance of objects in santonin-poisoning (due to violet blindness induced by the poison) or to red vision (erythropsia induced by fatigue of the retina for the short-waved rays of the violet side of the spectrum).

Illusions of hearing consist mostly of the reconstruction of words out of inarticulate sounds, or of the misinterpretation of the words or



sentences spoken in the patient's hearing. He may transform them into smoking, indecent, derogatory, or flattering words.

Illusions of common sensibility are, perhaps, more important in insanity than hallucinations of this sense. But they are difficult to study and establish. It is probable that the unsly, earthy taste of food often complained of by patients is more an illusion of touch than of taste.

Illusions of smell and taste are, in the main, unpleasant in character and are more common than hallucinations of these senses.

Illusions of organic sensation are frequently noted, and consist of such sensory metamorphoses, for instance, as the mistaking of intestinal motions for pregnancy, and the feeling of distention or increase in size of various organs (particularly noticeable in epilepsy and paresis).

Illusions of muscular sense or of movement are rare.

Illusions, like hallucinations, may form their material from the concepts at the moment in consciousness, or from latent memory-pictures.

The theory of the cause of illusions is analogous to that of hallucinations. They arise from a pathological recurrent influence of the excited memory-picture cells upon the sensory cells. The difference lies in the association, also, of an actual external stimulus which undergoes transformation.

Illusions are much more common than hallucinations, and are not seldom met with in normal persons. Often they are difficult to distinguish from one another. Sometimes it is impossible to differentiate true illusions from so-called illusory judgments, in which we are concerned not so much with a transformation of sensation, as with an erroneous judgment of the character of a normal sensation.

Illusions are noted in all forms of psychoses, especially in acute forms. They are particularly noteworthy in the hallucinatory form of paranoia.

**Disorders of Intensity of Sensation.**—These consist of hypesthesias, anesthetics, and hyperesthesias. Hypesthesias and anesthetics are observed in various psychoses which are complicated by such disorders as hysteria, chorea, multiple neuritis, tabes, focal cerebral lesions, etc. Hyperesthesia is also encountered in complicating disorders, such as hysteria, tubercular meningitis, neurasthenia; but is also often noted in the prodromal stages of many acute psychoses. It is especially remarkable in the insanities of childhood. A valuable objective sign of hyperesthesia is exaggeration of the superficial reflexes.

**Disorders of Sensory Tone.**—Agreeable or disagreeable feeling, associated with sensation, is described as sensory tone. Sensory tone may be perverted in insanity so that, for instance, fragrant is perceived as unpleasant, dissonance as pleasant, and vice versa. One notes such perversions in the slight psychopathic conditions of pregnancy in the form of capricious tastes and appetites. Homosexual perversion is a form of this disorder manifested in the domain of sexual sensation. Pathological disorders of the intensity of sensory tone consist of hypalgesia and analgesia, hyperalgesia, hypobolmia, and hyperbolmia.

The hypalgesias are noted in hysteria, tabes, congenital and acquired mental deficiency, and in severe hallucinatory confusion.

Hyperalgesia is observed under the same circumstances as hyperæsthesia. It is most often seen in hysterical and neurosthenic insanities, and almost exceptionally at certain points (such as the supraorbital, infraorbital, mental, Valsævi, iliac, intercostal, mammary, vertebral, and cranial suture points) pressure elicits pain. The pain of hunger, which leads in many psychoses to pathological hunger (bulimia) belongs in this category.

Hypæsthesia is a morbid diminution of the feeling of pleasure in any sensory perception. It is more important in the domain of sexual sensations than in others, where it may reach the degree of anhedonia. Sexual anhedonia is not uncommonly developed on the basis of a serious hereditary degeneracy, and is frequent, too, in organic disease of the central nervous system (tubes and paresthesia) as well as in toxic conditions (alcohol, cocaine, morphin). Hypæsthesia, in connection with hunger sensations, may reach the state of complete psychic anorexia in some insanities.

Hyperæsthesia is a morbid increase of positive sensory tone (agreeability of sensation), and is noted most often in relation to sexual sensations.

**Disorders of Memory-pictures or Ideas.**—Every stimulus in arousing a sensation in the cerebral cortex leaves some material vestige or impression, which remains as a latent memory-image or picture, latent presentation, or idea. Countless numbers of memory-pictures left by innumerable sensations of all kinds are stored away as a material deposit in the brain-cortex. These are reawakened either by the same or a similar stimulus, or excited through the stimulus of some idea-association. Only a few of the millions of memory-pictures are awakened to life at any one moment; all of the others remain latent. The general concept of any particular object is made up of the association of many centers in the brain, some of which are far apart, such as the smell, feel, taste, color, sound, and name of the object. The relation of this object to others of its kind is present in other associations, and these again in others, so that the material basis of an idea must be a perfect network of association fibers; and all of this labyrinth is connected with the complex series of language-centers, but particularly with the motor and auditory speech-centers, which are trained up from earliest infancy to associate the spoken word with the concrete conception. A word, therefore, expresses, like an algebraic  $x$ ,  $y$ , or  $z$ , some very intricate and complicated formula. Take words like "home," "right," "wrong," and so on, and think what a countless number of associated memory-pictures each one must represent! Words are simply convenient abbreviations which render more easy the use of concepts in idea-associations.

We distinguish in every idea four cardinal properties: (1) The contents or meaning; (2) distinctness; (3) associated affects; (4) energy or intensity.

The pathological disturbances of ideas may be studied under the headings of disorders of their evolution, durability, concomitant affects and associations.



**Defective Evolution of Ideas.**—The number of concepts stored up in the brain varies enormously under normal conditions with individuals and races. In method psychology we find the number of ideas extremely small among congenital defectives, such as the idiot, the imbecile, and the feeble-minded. The idiot may preserve rudimentary memory-pictures of the simplest things, such as food and eating, light, darkness, clothing, but without speech associations; he will have none of other persons or other objects about him. In the imbecile the concepts are more numerous and may be known by name; he recognizes persons and objects, distinguishes simple colors with difficulty, may have number concepts as high as ten; he has a few concrete ideas, but, as a rule, no abstract ideas. The feeble-minded has a larger number of memory-pictures, may have abstract ideas, recognizes the significance of likeness and similarity, and may use the words God, right, wrong, etc., but in reality be unable to tell the meaning of such complex, abstract conceptions. It is necessary, therefore, to avoid concluding that the idea is present because the word is spoken by such a patient, for it is particularly characteristic of the congenitally feeble-minded to be apt with words while deficient in grasp of their meaning.

**Disorders in Durability of Memory-pictures.**—The forgetting of a memory-picture, when the stimulus and sensation producing it are not repeated, may be considered to be due to its gradual erasure by the influence of the nutritive processes which affect the cortical ganglion-cells equally with all the elements and tissues of the body. This physiological destruction of the memory-picture is always very slow, but by pathological processes may be rendered enormously rapid. The destruction may be diffuse or limited to one sensory sphere (for example, apraxia, where the sensory ideas of objects are lost, though the sensory apparatus may be intact; mind-blindness, word-blindness, mind-deafness, word-deafness, etc.). But these limited defects of memory-pictures are due to focal lesions in the brain, and do not concern the alienist so much as the diffuse destruction of ideas, although it is true that the latter may sometimes be a sequel to a circumscribed lesion, and, on the other hand, that the diffuse disorder may, as in general paralysis, sometimes affect one region more than another. A loss of concrete ideas, such as general concepts of relationship, etc., which are represented by a wide-spread association network in the whole cortex, can only be caused by a diffuse, far-reaching disturbance. We see examples of such loss in the acquired dementias of paresis, epilepsy, and senility, dementias secondary to acute psychoses, and dementias due to toxic agents. It is natural that the latest memory acquisitions should be lost first, and the older memories successively later, in direct proportions to their age, according to a certain "law of regression," as Ribot terms it. This is to be explained by the want of permanence and stability in the newest arrangement or concatenation of protoplasmic molecules and ganglion-cells. The older impressions have become more fixed and durable.

Since an experience leaves behind not alone a single memory-picture, but a whole series arranged in chronological order, we may, as in amnesia, find pathological states in which there are losses of such series of ideas during a definite period of time. The so-called *subconscious*

or unconscious states are examples of this phenomenon. They are observed in epilepsy, intoxications, hysteria, sarcolepsy, hypnotism, somnambulism, injuries to the head, and in transitory insanity.

**Affective Disorders.**—Pleasurable or disagreeable feelings accompany ideas, just as they do common sensations; so that there is an intellectual affective tone analogous to sensory tone. If two ideas be presented simultaneously, and if one of these have a stronger emotional quality than the other, the tone of this will be irradiated to the other. Ziehen, in describing irradiation, gives the following example: "If I have met with an accident in any place, afterward not only is the memory of the injury accompanied by an unpleasant feeling, but the memory of the place is likewise mingled with a disagreeable affect. Furthermore, when I again see the spot where the accident occurred, I may feel again the sensation of the injury, accompanied by its unpleasant sensory tone." Here the memory-picture arouses the sensory tone of the sensation experienced. This is termed reflected tone. The most important consequence of the laws of emotional irradiation and reflection is that if in a certain period of time one or several sensations and ideas have a strong and similar emotional tone, all other sensations and ideas presented to the mind during the same period of time will be colored by the tone of the former. Such irradiation creates our moods, which are hence the abstract or summary of the similar emotional tones of the ideas and sensations experienced within any definite period of time.

Moods and emotions influence strongly the flow of our ideas, and, as a consequence, our actions. Depressed moods or affects inhibit, while exalted affects increase the flow of ideas, and likewise the resultant actions. Depressed affects are more durable and persistent than exalted affects. The latter subside rapidly. The more complicated ideas, such as justice, honor, law, family, patriotism, etc., are accompanied by a specific affect or tone which we designate as ethical feeling. Ethical feeling is the result of numerous irradiations, which the single idea acquires from all of the ideas associated with it; and the sum of the ethical feelings of an individual gives him his character (Ziehen).

In morbid psychology we classify changes in the affects as pathological depression, exaltation, irritability, apathy, and mutability.

**Depression.**—Depression is observed in many forms of insanity, particularly as a prodrome, but is characteristic of the melancholy types. It is a very common prodrome of acute mania, and a long period of morbid depression is frequently noted as an antecedent in general paresis. It is observed in neurasthenia, in hypochondriasis, and not seldom as an interlude in any psychosis. It is the cardinal symptom of melancholia. Depression is a mental consequence or accompaniment of sorrowful or dreadful hallucinations and ideas, and is, under such conditions, termed secondary. It is primary depression with which we are more concerned in insanity—a depression not at all or but slightly motivated by such hallucinations and ideas as we have just described, but a mood which takes possession of the mind of the patient and gives its own original color to every thought arising in his mind and to every external object presented to his consciousness. Past, present, and future are alike under the shadow of this mood. When mild in degree,



the patient feels only an inexplicable sadness—a certain restlessness or state of worry; but when extreme, this general mood of sadness becomes a condition of pathological anxiety—a mixed feeling of grief and dread, often accompanied by a feeling of suffocation or pain about the heart, and, therefore, frequently designated as “precordial anxiety” or “precordial fright.” When primary depression is present, the patient feels the change in his mental condition, observes that he no longer is cheered by the usual pleasant events of his daily life, that these rather intensify his misery. The affection and sympathy of his friends and family either awaken no response in his own breast when he tends to believe that he has lost all natural feeling, or they may awaken suspicion, dislike, and distrust. The inhibition of the flow of thought restricts his ideas to himself and to the somber contents of his mind. He is not easily distracted from such contemplation, and answers questions, if at all, very slowly and with great difficulty. Nearly all cases with morbid depression complain of disorders of visceral sensibility, from a slight sense of constriction at the throat to precordial distress, from a general feeling of illness and uneasiness to a feeling of extreme and general restlessness. No doubt depression influences often the entire innervation of the body, so that the patient wrings his hands, picks his fingers or head, walks up and down, is extremely agitated, goes into a condition of cataplexy or catamnia, or, on the other hand, remains absolutely immobile and requires the service of others for every movement. The muscles of the peripheral arteries contract and increase the frequency of the heart's action. The constriction of the throat is probably an actual contraction of the oesophageal muscles. Precordial anxiety is most likely due to vasomotor disturbance in the vessels of the heart. The constipation so frequent in depressed conditions depends doubtless upon retardation of peristalsis. Thus we observe in one case motor inhibition, in another motor excitement, and in some alternations between the two.

In seeking to explain the mood of sadness and uneasiness which he feels, the patient tends to develop delusions. He invokes the first ideas which would naturally come to him under such circumstances. He seeks in his past life for some sin, the commission of which may have brought this punishment. He magnifies some trivial error in his youth into an unpardonable sin. Or he comes to think that poverty stares him in the face, or that he can never recover from an insurable illness which has taken possession of him. Occasionally, a persecutory delusion is evolved from a primary depression.

*Excitation.*—Excitation is occasionally noted as an intercurrent symptom in any psychosis. It sometimes alternates with depression, forming a constant cycle, as in circular insanity, and sometimes it presents itself during convalescence from melancholia as a reactive phenomenon. In the majority of cases of general paresis a period of exaltation develops. In maniacal states, however, it is observed as a cardinal symptom. As with depression, we distinguish a secondary exaltation consequent upon agitated hallucinations and ideas, and a primary or unmotivated exaltation. In exalted moods the æsthetic sensations are pleasurable and give rise to feelings of perfect health.

strength, and vitality. The stream of ideas is hastened, and as a result the patient becomes, according to the degree of exaltation, talkative and garrulous, or exhibits a veritable logorrhea,—a constant, rapid flow of words,—which may often assume a rhyming, singing, or oratorical character, with marked incoherence. The rapid stream of presentations is paralleled in the motor sphere by increased muscular activity, varying from busy occupation with nothing to gesticulating, grimacing, and dancing, and to the wildest and most violent motor excitement.

Primary exaltation frequently gives rise to delusions of a grandiose character, though these are unstable and fleeting, corresponding to the rapidity of change in the contents of consciousness. But the feeling of well-being and of egotism which makes up the fundamental mood of the exalted patient leads him to be extremely impatient of any restraint of his activities; and, in consequence of this, the reactive feeling of aggressive anger and fury is easily aroused, leading to acts of violence and destruction.

*Irritability.*—Irritability is a condition which has to do chiefly with the affects of anger and rage. While observed in association with exaltation, as just noted, and among the prodromata of various insanities, it is particularly characteristic as a primary emotional state of congenital and acquired mental weakness, neurasthenic insanity, and the epileptic psychoses. In the latter it not infrequently becomes a true *jeu epileptique*. Irritability is occasionally noted in the convalescence from acute insanities, sometimes conjoined with a peculiar tearfulness, a lacrymose irritability. While most of the affects of both depression and exaltation are concerned with the ego, the affect of anger differs markedly from these in that it has to do with persons or objects outside of one's self. At the same time anger is a depressed emotion, but with certain peculiarities. In its influence upon the flow of ideas and upon action it first retards or inhibits, but finally, by an accumulation of stimuli, induces a sudden motor explosion, which may vary from simple aggressiveness to the most uncontrollable fury. Abbreviation of the usual play of motives is characteristic of the motor explosions of anger and fury. The sensory stimulus is carried directly into the motor arena, without the intercession of ideas or inhibitions, which accounts for the frequent occurrence of outbreaks of violence and destructiveness, followed by complete or partial amnesia as to the acts perpetrated.

Diminution or cessation of sensory and intellectual emotional tone gives rise to the condition known as partial or general apathy. A general apathy is frequently observed in neurasthenic insanity and in stuporous states, but it is more common in certain cases of melancholia. Such patients will complain, paradoxically as it may seem, of a painful feeling of having lost all feeling. They say that they feel no affection for their children, no hope of getting well, no pleasure in anything, no grief at the loss of friends, that their hearts are turned to stone. Sometimes ordinary sensory feeling seems absent also, and they say they can feel neither heat nor cold, nor the pain of a cut or injury. One must distinguish between an apparent apathy and a want of atten-



tion consequent upon self-centering of the thoughts on strong delusions and hallucinations.

Partial apathy or limited defects of the emotions, as well as of special and cutlinary sensation, are frequently encountered in various grades of congenital idiosy and acquired mental weakness. Defects of the higher forms of intellectual sensory tone, the ethical feelings, which we meet with in some of these cases, constitute the so-called moral insanity.

In certain psychoses a general apathy may be so great and the horizon of intellectual processes so narrowed that the condition amounts to a pseudodementia (Magna), though there is truly no actual defect of intelligence, the mental functions being merely temporarily inhibited or suspended.

A peculiar mutability or lability of affects is not an infrequent phenomenon in insanity. Laughing and crying at the same time is not a rarity in persons who are not insane, being the result of the commingling of pleasant and distressing ideas present at the same moment in consciousness. The emotional pendulum swings quickly from one extreme to the other. Such disequilibrium is particularly characteristic of hysteria, and is notable in the hysterical psychoses. But irritability and rapid alternation of cheerful and pathetic affects are also encountered in the most various psychoses. The chronic melancholic with his sad face and automatically repeating his set phrase, "I am going to be killed," may laugh out suddenly at a funny incident and immediately relapse into his habitual mental attitude. In the same manner the paranoiac may forget momentarily his persecutory delusion. In general paresis this swinging from one emotion to the other in the most rapid manner is extremely characteristic. Mutability of affects is indeed most common in combination with conditions of intellectual defect or mental weakness.

#### DISORDERS OF THE IDEA-ASSOCIATIONS.

An idea-association is a psychological series, beginning with a stimulus and ending with a movement, between which may be one or two or more memory-pictures, some coming into consciousness, others remaining latent, but all associated by the nerve-fibers running between the ganglion-cells of the cortex in which are deposited the sensory impressions. The selection and serial course of ideas in the stream of thought are determined by fixed laws. One of these is the law of similarity-association—*i. e.*, a sensation induces an idea (seeing a flower gives the idea of a flower) and another latent idea is aroused by this (a rose) because the second memory-picture has marked similarity to the first idea—the rose is remembered or recognized. Every recognition contains a judgment, since a new sensation is seen to be like a former sensation.

Another law is that of simultaneity of reception—*i. e.*, memory-pictures are associated when their sensory stimuli have been received at the same time. For example, the sight of a friend recalls the city, the street, the house where one first saw him, and many others in a highly

complex series of associations. Not all of these, however, will arise at sight of him. Perhaps it may be one or two, perhaps others; so that another factor arises—viz., the degree of associative relationship. Still another factor is the feeling (the intellectual sensory tone, the affect) combined with each of the memory-pictures. Those memory-pictures will rise soonest into consciousness which are combined with the liveliest emotions, agreeable or disagreeable, pleasant or painful. Ideas with strong affects have a greater chance in the conflict of ideas to rise up from their latency into consciousness. Still another feature of this scheme is that the latent ideas with their numerous associations influence one another reciprocally, some to excite and some to suppress or inhibit. While simpler ideas are arranged in a sort of serial association one after the other, on a higher plane the successive memory-pictures are bound together into judgments and conclusions. Ziehen cites the example of the simple judgment, "The rose is beautiful," in which we have not three ideas discreetly ranged one after the other, but the ideas "rose," "is," and "beautiful" stand in a thorough relation to one another. This form of idea-association is designated as a judgment-association.

The normal stream of ideas, or idea-association, has a definite swiftness which varies in different individuals and in the same individual at different times. In psychopathology we learn that agreeable or pleasant affects lessen and disagreeable or unpleasant affects retard the flow of thoughts.

The pathological disorders of the idea-association are to be classified as follows:

1. Disorders of memory.
2. Disorders of attention.
3. Accelerated flow of ideas.
4. Diminished flow of ideas.
5. Disturbance of the connections between the ideas of the idea-association (incoherence).
6. Falsification of the judgment-associations (delusions and imperative ideas).
7. Defective judgment-associations (weakness of judgment).

**Disorders of Memory.**—Recollection according to the principle of similarity-association is the calling up (by a sensation) of a memory-picture of earlier, similar, or identical sensations.

Recollection is disordered or destroyed by loss of the necessary memory-pictures, by any general marked retardation of cortical associations, and by dissociation of the idea-association.

Dissociation is equivalent to incoherence, and when a general incoherence exists, disorder of recollection is the rule. The patient then confounds persons and objects, and often loses the ideas of place and time (a condition for which *dissociation* is the best name). The peculiar *parannesia* observed in alcoholic psychoses, especially in the delirium accompanying alcoholic neuritis, is a striking example of this loss of orientation. The mistaking or confounding of persons and things depends upon illusions, delusions, incoherence of ideas, lack of distinctness of the requisite memory-pictures, or, finally, upon voluntary



caprices of the patient. In alcoholic paranoia and epileptic insanities, and sometimes in other psychoses, we encounter the so-called "hallucinations of memory"—a bad term for the phenomenon experienced sometimes by normal individuals, of having seen (his or that thing, or of having been in the same place before, although in fact the object and place are absolutely new.

From the practical standpoint it is wise to investigate two conditions in relation to memory: first, the memory-store, made up of all past experience; and, secondly, the patient's power of adding new experiences to this memory-store (power of retention, *verförmigheit*). Amnesia is a defect in the memory-store, often sharply circumscribed as to time-relations by some trauma, accident, emotional shock, or fit (a classic symptom, for instance, in epilepsy). Amnesia may be total or partial. The so-called "summary remembrance" is a kind of amnesia in which there are defects here and there in the memory-store clustered about some critical period, some of the experiences being intact and others destroyed. When the amnesia extends not only over some critical period (such as the time of an accident, fit, trauma, fright, etc.), but backward far beyond the period in question, for days, months, or years, it is called retrograde or retroactive amnesia.

**Disorders of Attention.**—Condilliac stated that if amid a multitude of sensations there is one which predominates by its intensity, it is thereby transferred into attention. Ribot<sup>1</sup> regards spontaneous attention as always caused by emotional states. The writer believes, with Ziehen, that attention is never voluntary, but always spontaneous; that it is the awakening of one idea from the impressions of the innumerable sensations impinging on our sensory surfaces. Such attention depends upon several factors. One is intensity. Another is correspondence of the received sensation with some latent memory-picture. A third factor is the affective quality, or sensory tone of the sensation. A fourth factor is the combination of latent ideas.

The disorders of attention are morbid diminution and morbid increase. The former is extreme in idiots, and noticeably in patients dominated by strong hallucinations or overpowering delusions. By pathological increase of attention is meant the crowding of numerous sensations and ideas into consciousness, such as is observed, for instance, in manical states.

**Accelerated Flow of Ideas.**—In the highest degree of pathological increase in the stream of thought we observe not only a rapid recombination of the associated ideas, but their swift transfer to the cortical motor areas, so that gesticulation, logorrhea, and motor agitation become strikingly prominent. It is an ideomotor excitement. It may be so severe as to present a secondary incoherence. In moderate degrees of acceleration the words spoken by the patient may, by their sound, arouse associations, so that we observe in the speech of the patient a tendency to rhyming associations and Verblagerung. The almost constant combination of augmented flow of thought with an exalted and cheerful mood is interesting and, at the same time, difficult to explain. Some believe that the exaltation is due to the patient's feeling of great facility

<sup>1</sup> "The Psychology of Attention."

and fecundity of thought. Others, again, consider the exaltation as the primary phenomenon, and that, as in normal individuals, the exaltation induces the free play of ideas. But it is probable that the cheerful mood and accelerated flight of ideas are simultaneous manifestations of the morbid process.

**Diminished Flow of Ideas.**—In this symptom we have features quite opposite to those manifested in ideomotor excitement. In the place of increased we have diminished attention to the sensory stimulus, and retarded transfer of the awakened ideas-associations to the motor areas (motor inhibition). In any noteworthy inhibition of the flow of thought we observe also difficult and retarded recollection and more or less complete cessation of all voluntary movement. Speech becomes slow, the patient seeking laboriously for words, and these are simply whispered, not spoken aloud. In severe degrees only slight movements of the lips are made, or complete mutism is presented. Sometimes a word or phrase will be repeated monotonously over and over; a single motion of the arm or body may be reiterated for hours (stereotyped movements). The general musculature of the body may be completely relaxed and flaccid (motor-inhibition with resolution) or in a state of tension (catatonic inhibition), or in the condition known as *flexibilitas cerea*. The condition designated as stupor comprises three cardinal symptoms—*viz.*, diminished attention, thought-inhibition, and motor-inhibition. Stupor may be primary or secondary. When secondary, it is ordinarily induced by hallucinations of ecstatic, dreadful, or imperative nature. Stupor from ecstatic hallucinations is frequent in hysteria and epilepsy, and from dreadful hallucinations in melancholia (catatonic syndrome). Primary stupor is another name for primary dementia.

Depression with thought-inhibition is common, and among the depressed affects associated with it we observe most frequently anxiety. According to the motor symptoms prominent in such cases, such as flaccidity (or resolution), catatonic rigidity, and restlessness, we distinguish three types—*viz.*, melancholia passiva, melancholia attonita, and melancholia agitata. The usual motor inhibition is concealed in melancholia agitata by the expression movements of anguish, such as wringing the hands; picking the fingers, face, or scalp; restlessly moving to and fro, anteroposterior or lateral oscillations of the body, and the like.

In the diagnosis of thought-inhibition we must be careful to distinguish, in the first place, actual defects of intelligence or conditions of dementia. Then we must distinguish the primary form without affects and with affects, and the form secondary to hallucinations and delusions. Some of the diagnostic criteria are:

Dementia and others are stationary or progressive conditions, while, on the other hand, in thought-inhibition there are transitory variations—intervals of diminished inhibition.

Thought-inhibition is almost always combined with motor-inhibition, while this latter symptom is not observed in defects of intelligence.

The judgment-associations in defective intelligence are also defective, and wrong answers are often given to questions. This is not true of



states of thought-inhibition, where correct answers are generally made, if made at all.

**Incoherence.**—Incoherence is a dissociation of serially related ideas. Such dissociation may involve also the sensations which arouse a series of ideas and the motor sequence of a series of ideas. In a complete general incoherence, then, the patient recognizes neither persons nor objects, calls everything by its wrong name (*pseudoparaplasia*), uses everything wrongly (*pseudo-apraxia*), answers questions with absolute irrelevancy, and shows even incoordination and *pseudo-ataxia* in his movements. When the incoherence is marked in the sensory perceptions, we speak of lack of orientation; it was formerly termed a disorder of self-consciousness. When the motor incoordination is extreme, it may amount to veritable jactitation and *pseudochorea*. Incoherence is most remarkable, however, in the speech, writing, and mimetic expression of the patient. The gestures and facial movements have no relation to the contents of consciousness; laughter may accompany dreadful hallucinations and a fearful countenance some jocular idea. As regards speech, if the incoherence is of mild degree, only the sentences are misplaced; if of severe degree, the very words in the sentence are jumbled together, and we observe the phenomenon of *verbigeneration* and the manufacture of new words. The handwriting of the patient may present the same incoherence as the speech. The term *confusional insanity* has been used to describe the form in which the symptoms are want of orientation, incoherence of ideas, and motor incoherence. Incoherence may be primary or secondary, generally the latter. As a primary phenomenon, it is the cardinal symptom of the incoherent form of *paranoia*. Secondary incoherence is due to extreme rigidity of the stream of ideas, to accumulation of rapidly changing delusions and hallucinations, to strong depressing affects, and finally to actual defect of intelligence. It is often difficult to distinguish primary from secondary incoherence, and far from easy to differentiate the causes of the latter.

**Delusions and Imperative Ideas.**—Ideas are associated with judgments as to similarity, simultaneity, properties of objects, etc., and such judgment may be correct or erroneous in normal individuals, according to the weakness or strength of judgment, and according to the degree of correspondence between the sensory perceptions and the objects or events of the external world. The normal mind, however, generally corrects its errors of judgment by repeated experience and better education—a physiological process. The pathological errors of judgment are the delusions of the insane. These delusions are usually judgments founded upon incorrect sensory impressions, such as illusions and hallucinations. They are rarely corrected by experience, as is the case with physiological error. But there are many cases in which a definite boundary-line can not be drawn between the delusions of the sane and those of the insane, as, for instance, in the delusions of the superstitious and of spiritualists.

The delusion is the most frequent form of pathological error of judgment, but the imperative idea is also a pathological error of judgment, though less commonly met with. Delusions are seldom influenced by, or, in fact, associated with, attempts at correction by the judgment;

whereas imperative ideas are usually recognized as such by the patient, but force themselves into consciousness despite the efforts of the judgment to dislodge them.

A delusion may arise in the mind as a primary idea without an interested sensory basis, in the same way as an imperative idea. It may be a logical deduction from other delusions, or, as already stated, be the product of illusions or hallucinations. It may be the result of a dream carried over by weakened judgment into the waking life. It may develop, as in melancholia or mania, from the attempts of a patient to explain the origin of his depression or exaltation. Thus, the melancholic believes that his suffering must be due to his bad conscience, to some sin that he has committed, to some serious disease of his viscera, and the like. The patient with exaltation of his emotional life develops expansive ideas as to his strength, beauty, intellect, wealth, position, and so on. The character of delusions developed in the insane is as multi-form as are the ideas in the mind of man.

Depressive delusions are almost always connected with the idea of having committed a sin, of having some disease (hypochondriasis), of having lost all property, or of persecution. Contrasted or antagonistic delusions of grandeur are sometimes observed at the same time in connection with depressive delusions. Thus, one patient, while weeping and wringing her hands, told me she was the queen of the world, but was unable to do her duty because she did not know all languages. Ziehen tells of a patient who said, "I was the Holy Ghost. Had I used my omnipotence, we would all be happy now. But I am cursed. I have killed the Holy Ghost. The whole world is in misery and dread through me." Hypochondriacal delusions generally arise from disorders of common or organic sensibility, conesthetic sensory impressions, though they also develop from attempts at explanation of a depressed mood and from hallucinations. The patient is certain he has cancer, consumption, syphilis, brain-softening; that he is impotent; that his alimentary canal is closed up; that his brain has been removed; that his viscera and tissues have been metamorphosed into stone, glass, wood, and the like. A peculiar form of hypochondriacal delusion is the so-called micromania not infrequently observed in depressed periods of general paresis. Patients with micromania assert that whole viscera have been removed from their bodies, that their blood is all gone, and then they have been reduced in size. Thus, one patient told me she was so small she could be put into a pill-box. Another said his intestines were absolutely closed up and he should have to be cut open to have the obstructions removed. The delusion of pregnancy arises from perversion of abdominal sensory impressions.

The delusion of persecution differs from the other depressive delusions in that it has to do with the enmity of other persons in the environment, whereas these are concerned altogether with the ego of the patient, his own conscience, his own mind, his own body. The delusion of persecution is important to the general practitioner, because it is very common, because it is met with so often outside of institutions, because it not infrequently leads to assaults and murder, and because its significance in prognosis varies with the species of mental disorder in which it



is encountered. It is observed, for instance, in toxic insanities which are curable; in melancholia, in which cure is difficult; and in paranoia, which is incurable. The most common origin of the delusion of persecution is from hallucinations. The patient hears mocking or threatening voices, he tastes poisons in his food, he sees lowering looks and menacing gestures, he feels singular sensations in his body which must be due to irritant poisons thrown upon him or to electricity, or he smells noxious gases. The delusion of persecution may grow out of a series of hypochondriacal delusions, in the attempt of the patient to explain the origin of his miseries. It may arise also from the delusion of having committed a sin or crime, the patient imagining that every one hates him and follows him to punish him. Sometimes these persecutory delusions are referred to the influence of unseen agencies—hypnotism, telepathy, electricity, magnetism. Sometimes they have to do with the property or social position of the patient; he believes his belongings are being stolen, or his character maligned. Sometimes erotic ideas are bound up with persecutory ideas; a woman believes herself secretly cohabited with at night, or even by day, through occult means; a man thinks he is made impotent, that his seminal fluid is being drawn off. Obviously, these latter ideas often rest upon perverted sensory impressions received from the sexual organs. In seeking to discover the origin of the persecution, the patient often at first settles upon some one definite individual, but later, when he finds the methods of persecution innumerable and that his enemies follow him wherever he goes, he can not believe that any one person could do so much; he reaches the conclusion that it must be a wide-spread conspiracy, such as could be carried out only by some large affiliation of persons, such as societies of Freemasons, anarchists, Jesuits, lawyers, and police. The delusion of persecution occasionally develops from a delusion of grandeur; the patient believes he is persecuted because of his wealth or exalted position. More often, however, the contrary is the case, the patient coming to believe himself some extraordinary personage because of the persecutions to which he is subjected.

Another interesting form of depressed delusion is that of negation (*délire de négation générale*), which has its origin usually from an idea of having sinned. The patient thinks he must be the devil himself, his sin is so great; consequently he can never die, he must suffer forever; then, with the growing idea of the enormity of his sin, he comes to believe that God and mankind and the world exist no more.

Delusions of grandeur vary from simple, expansive ideas of the patient's importance, prerogatives, and powers, to delusions of being inventors, geniuses, prophets, reformers, titled and royal personages, and even Christ, God, and the mother of God. Besides his own personality, his environment may be rested with grandiose qualities—his room a palace, his stove but a crown, pebbles diamonds, his children possessions, and so on. A peculiarity of the ideas of grandeur observed in general paresis, which is quite pathognomonic, is their enormity or, rather, monstrosity. It is not enough to be wealthy, but sextillions of pounds can not hold the gold and jewels. It is not sufficient to have a dozen children, but billions of children are given birth to nightly by his

innumerable wives. He will make a new Niagara, by bringing the Pacific Ocean over the Andes. Should sexual ideas prevail, he may say that his penis is a mile long, and his testicles are large diamonds. He will move the asylum across the United States on a road of solid gold. Such enormities betoken great weakening of the intellect and judgment.

Primary delusions conduce more to fixity than delusions secondary to hallucinations. The latter, depending as they do upon the stability or instability of the morbid sensory impressions, change with these. When delusions become fixed, they tend to crystallize or become systematized. Systematization consists of combining with the fixed idea complementary delusions in a more or less logical order or of the fantastic elaboration of the original delusion. The degree of organization and perfection of the delusional structure will depend upon fancy, logical faculty, social position, and education of the patient. The most common form of systematization is in the development of secondary grandiose ideas upon a persecutory basis. But almost any of the depressed and excited delusions previously described may become fixed, systematized, and permanent through the life of the patient.

Delusions may have a retroactive effect in awakening sensory impressions, instead of being aroused by them—that is, may induce illusions and hallucinations. For example, the persecuted patient perceives voices, odors, tastes, pains, etc., often because of his mind being in a state of expectant attention.

**Imperative ideas** force themselves into consciousness in spite of the efforts of the patient—who recognizes their morbid character—to correct them. They are accompanied, almost without exception, by a depressive affect, a painful sensory tone. They are extremely common in neurasthenia. Senseless phrases or doggerel repeat themselves over and over in the patient's mind. The many varieties of phobia are familiar examples of imperative ideas in neurothénics (agoraphobia, claustrophobia, mysophobia, etc.). Imperative ideas are also observed in melancholia and in a form of insanity which has been designated as insanity from imperative ideas. In very rare instances they are encountered in early stages of general paresis. They always develop on the basis of a congenital or acquired neuropathic or psychopathic constitution, and are apt to become obstinate features in the mental organization. Almost every imperative idea has its inception in some sort of sensory impression, and the idea may lead to compulsory actions on the part of the patient. But between the imperative idea and the consequent action there is generally a play of judgment, a faltering between the imperative idea and antagonistic or inhibiting concepts. For instance, the patient feels a compulsion to lock a door which he feels sure he has already locked. After an inward debate as to whether he should go back and assure himself that it is locked, which may last many minutes or longer, he goes to lock it, and on leaving the door again the imperative idea arises that it is not locked. The same play of antithetic ideas may occur in reference to anything—the addressing of a letter, the return of a book to a shelf, acts of dressing and undressing, the crossing of a street, etc. In some cases the imperative



idea takes the form of compulsion to jump from a height, to laugh in unseasonably places; or obscene and sacrilegious words, sentences, and fancies may thrust themselves obstinately into the consciousness. For example, a gentleman, and a good Christian, came to me recently overwhelmed with the sacrilegious conceptions which first came to him at a church-service a week or two before—ideas of cohabitation with the Virgin Mary and filthy expressions in relation to Christ. A lady consulted me about a morbid fear that she had of caviary birds. She could not enter a house or hotel in which there was a caviary bird, because she was afraid that bird-wood might get about and in some way get into her mouth, be swallowed, and grow in her stomach. The contents of these impulsive concepts are as varied as those of delusions, though they are almost, without exception, trivial or unpleasant.

*Folie du doute* is a form of mental disorder in which compulsory ideas assert themselves in the form of questions, religious, metaphysical, or in regard to the most trivial things or events (Shall I do this or that? Why is the table round? Why is the chair by the bed? Why are two and two four?). One young lady is so incapable of deciding any question that comes up in her mind that she does not know whether she ought to dress or undress, go to bed, eat, sleep, pray, or consult a doctor. Every trivial question of the day requires hours of painful and agonizing debate in her mind.

Impulsive ideas frequently impel to compulsory speech and actions. Coprolalia is a not uncommon form of impulsive speech in which the patient is impelled to the utterance of obscene words. Quite undogmatically the patient may be made to make grimaces, or may develop the so-called *maladie des fies*.

**Weakness of Judgment.**—Innumerable memory-pictures and associated ideas take part in the process of comparison and decision which we know as judgment. Hence any disorder of memory and of its associations, such as loss, defect, or perversions (delusions, hallucinations, or illusions), must naturally influence the character of the judgment. One of the common conditions which impairs judgment is, therefore, intellectual defect, such as congenital or acquired mental weakness. The criteria of idiosyncrasy and dementia are poverty of ideas and idea-associations and weakness of judgment. When delusions or impulsive ideas exist, the crises of judgment are due to the overriding and eclipsing by single ideas and idea-associations of all others which would in the normal mind give balance, control, and revision to the judgment. Defective judgment varies in degree from a slight loss of the critical faculty to complete deficiency. When the judgment is markedly defective, it depends upon actual organic changes in the brain, such as we observe in illness, terminal dementia, senile dementia, and general paresis, and hence as a symptom it is far more ominous than delusions and impulsive ideas, which usually rest upon a functional pathological basis. Its significance, then, demands a careful differentiation of this symptom from others with which it might be confused, such as incoherence and thought-inhibition. In incoherence the threads of thought are constantly lost. In thought-inhibition there are a depressive affect and extraordinary slowness of association with

correspondingly tardy answers, and, besides, there are variations of depth of inhibition, so that at times complicated answers and judgments are readily given. In actual weakness of judgment the judgments rendered are false, and the more incorrect, the more complicated the questions.

#### DISORDERS OF ACTIONS.

The actions or conduct of a patient depend directly and necessarily upon pathological elements in some part of the psychological processes—sensation, memory-pictures, idea-associations, and their emotional affects. They may be classified, following Ziehen, as—

1. Actions induced by sensory disorders.
2. Actions induced by disorders of memory.
3. Actions induced by disorders of the emotions.
4. Actions induced by disorders of the idea-association.

**Actions Induced by Sensory Disorders.**—Hallucinations and illusions affect the conduct of a patient often markedly, and their influence is always greater than that of normal sensations. Their dominance is the greater in proportion to their number and to the rapidity of their accumulation. Hallucinations gathered slowly in the course of weeks or months, while they may not be corrected, are at least subject to a certain amount of control by the inhibition of normal ideas. In the most chronic forms of hallucination the voices, common sensations, and visions tend to be ignored and to influence to a very slight degree the conduct of the patient. A very important practical feature in regard to hallucinations and their effects upon conduct is their uncertainty. They are never to be reckoned with, and one can never know what sudden violence or destructiveness may result from new hallucinations rising in the patient's brain.

**Actions Induced by Defects of Memory.**—These are observed in congenital or acquired weak-mindedness, where the conduct is directly ordered by sensory impressions, without that intervention of the play of motives which we observe in normal individuals. They are more like the actions of the lower animals, which may be complete enough in their way, but are not motivated by complicated abstract conceptions, because these are wanting.

**Actions Induced by Disorders of the Emotions.**—As already elsewhere intimated, simple depressed emotions are accompanied by a general motor inhibition, and simple exalted emotions by a general motor agitation. But when the depressed affect attains to the degree of anxious dread, we may have a restlessness, a desire for flight, which in itself amounts to a motor agitation. This anxious state often leads to suicidal attempts, and even to homicidal assaults, arson, and other forms of crime and violence. The whole nervous system seems to be in such a state of tension that only an explosion can give relief.

In apathetic conditions action is reduced to its minimum.

Where the higher affects, which are at the basis of ethical concepts, are absent or lost, as in congenital or acquired states of mental weakness, crimes against person and property are common.

In conditions of anger and rage there is at first a brief period of



speechlessness and immobility, followed by an explosion of blind and violent motor excitement, in which the most dangerous assaults may be made.

In conditions of changeability or lability of the emotions, we observe analogous motor states—sudden changes from weeping and wailing to boisterous cheerfulness, and vice versa.

A study of emotional expression is of particular diagnostic value in insanity, but the features of such expression and gesticulation are so well known that they need no detailed description here. Each mood, be it simple depression, anxious terror, excitation, anger, apathy, or emotional lability, has its own familiar motor habitments.

**Actions Induced by Disorders of the Idea-association or Stream of Thought.**—Under this heading are gathered the multiform modes of action caused by increase in the flow of ideas, retardation of the stream of thought, incoherence, delusions, imperative ideas, and weakness of judgment.

In increased rapidity of the flow of ideas we note motor agitation or morbid impulse to movement, varying from simple talkativeness, with active play of expression, to loud garrulity, grimaces, gesticulation, busy walking about, running, dancing, and, in extreme degree, to undressing, destructiveness of clothing, bedding, furniture, and blind throwing about of the body in every conceivable way. This so-called primary motor agitation should be distinguished from the motor agitation which is secondary to crowding hallucinations (hallucinatory agitation) and to emotions like terror and anger (affective agitation).

The behavior of the movements in regard to retarded flow of thought has already been briefly alluded to. There is a general motor inhibition, varying from simple slowness and difficulty of executing any movement, whether of speech or other muscles, to a complete cessation of voluntary movements, a stuporous or catatonic condition, in which the muscles may be absolutely at rest and flaccid or, on the other hand, in a condition of catonic tension. In true catonic tension every attempt at passive movement is resisted, but in another form of this there is a waxy flexibility of the muscles, so that the limbs yield readily to any passive motion, remaining in whatever position the physician desires to place them. Occasionally one encounters in cases of retarded idea-associations, as an expression of motor inhibition, a tendency to the repetition of some restricted voluntary movement in a rhythmical, stereotyped way for days, weeks, months at a time. Such stereotyped motions may be simple antero-posterior oscillations, lateral oscillations, whirling, walking to and fro or in a circle, waving the hands rhythmically—forms of ties exceedingly common in idiosy and imbecility, but common enough in melancholia and terminal dementia. The repetition of stereotyped or automatic phrases is analogous in character to such morbid movements. Motor inhibition is primary or secondary. The primary form is generally a simple resolution or flaccidity, occasionally a slight catonic tension or *stribilitus cerei*. Secondary motor inhibition is due to hallucinations, delusions, and states of mental weakness.

Incoherence of ideas leads to a dissociation also in the motor expressions of ideas, parapraxia, perimimia, incoördination, pseudo-ataxia, incoherent agitation, chorea magna, and jactitation. Such motor agitation may be primary or may be the secondary result of insupportable clashing hallucinations and delusions, rapidity of the flight of ideas or of intellectual defects.

Grandiose delusions exert their own peculiar influence on the demeanour and speech of the patient, according to the contents of the exalted ideas. We observe the proud bearing; the self-sufficient, haughty, or secret smile; the withdrawing from others; the tendency to deification of the person; the attempts to act the parts of the personage he imagines himself to be; the striking peculiarities of handwriting. In some instances delusions of grandeur lead to homicidal, rarely suicidal, attempts (self-crucifixion with the delusion of being Christ). Grandiose erotic ideas sometimes occasion masturbation. Coprophagy and other filthy habits may depend upon grandiose delusions as to extraordinary virtues of the patient's excretions.

In depressed delusions, particularly as regards ideas of sin and poverty, we observe the characteristic melancholy facial expression and attitudes. Attempts at suicide are frequent, and sometimes self-mutilation. Abstinence from food is especially common with the delusion of poverty, the patient feeling that he can not pay for anything.

Hypochondriacal ideas influence markedly the patient's actions and conduct. The hypochondriac may neglect every duty in the constant contemplation of his symptoms. He reads medical books, goes from one physician to another, takes to his bed perhaps permanently, and so on. The effects of hypochondriasis on motor functions are frequently remarkable, leading sometimes to astasia or abasia, or both; to hypochondriacal ataxia, tremor, or convulsive movements of the extremities. These hypochondriacal motor conditions are always the result of a series of morbid judgments on a hypochondriacal basis, and are to be distinguished from similar hysterical states which have an autocathomous origin without any antecedent conscious reasoning process.

The persecutory delusions lead to systems of self-protection of the most varied kind. Barricades, stopping up of cracks and keyholes, the wearing of peculiar clothing (silk, paper, etc., for instance, as a guard against electrical shocks), avoiding of food and drink which are suspected of containing poison, arming with weapons, frequent change of servants or residence, and complaints to the police or judicial authorities. Homicide is common in these cases.

Imperative ideas lead to imperative movements and actions, and generally in spite of the well-preserved consciousness and judgment of the patient. Such imperative actions are as various in character as the imperative ideas to which they correspond.<sup>1</sup>

**Accompanying Physical Disorders in Insanity.**—Among the

<sup>1</sup> The foregoing account of the psychopathology of insanity is largely a presentation of the views of Ziehen, to whose excellent work the author must refer readers for greater detail.



many somatic symptoms which may complicate or accompany psychoses are chiefly to be mentioned the following:

1. Motor disorders.
2. Sensory disorders.
3. Reflex disorders.
4. Trophic disorders.
5. Secretory and excretory disorders.
6. Temperature disorders.
7. Vascular disorders.

**Motor Disorders.**—These may be manifested in the form of morbid movements or paralysis. In the first category are assembled such symptoms as epilepsy, convulsions, chorea, choreiform movements, tremor, tic, staxia, masticatory spasm, and the like. The following table, modified from Ziehen, gives a general summary of the paralytic symptoms noted in insanity:

FORM OF PARALYSIS.	CHARACTER.	TROPHIC DISTURBANCES.	SENSITIVITY OR FLACCIDITY.	SENSORY DISORDERS.	FORM DISORDERS.
Hypochondriacal.	Usually limited to a certain form of movement.	No atrophy.	Flaccidity.	None.	Normal.
Hysterical.	Hemiplegia, hemiplegia, or paraplegia.	Dense atrophy.	Frequently contractures.	Hemianesthesia, etc.	Normal or hypertypical.
Cortical.	Hemiplegia or hemiplegia.	Dense atrophy.	Rigidity, contractures, local spasms.	Paresthesias, occasionally anesthetics.	Exaggerated usually.
Pyramidal tract.	Hemiplegia or paraplegia.	Dense atrophy.	Spasticity, contractures frequently.	Occasionally anesthesia, hemianesthesia, etc.	Exaggerated.
Peripheral.	Multiple or single.	Tricerebral with degenerative reaction.	Flaccidity.	Hypesthesia, stocking and glove areas of anesthesia often.	Lost.

**Sensory Disorders.**—Anesthesia and hyperesthesia have already been mentioned, but hyperalgesias and paresthesias of divers kinds are encountered among the psychoses, such as headache, migraine, neuralgias, feeling of fullness in the head, scotomata, tinnitus aurium, and so on. Neuralgia is occasionally a cause of insanity. Migraine is a frequent precursor of general paresis and concomitant of epilepsy. Lightning pains are noted in tabic types of dementia paralytica. Neuromathetic pains and paresthesias in the extremities, spine, and head are found in

neurasthenic forms of insanity. Where hysteria complicates a psychosis, there are often observed the sensory disturbances characteristic of that anomaly.

**Reflex Disorders.**—Changes in the reflexes are important in but a few forms of insanity. In paralytic dementia we observe nearly always exaggerated tendon-reflexes, but in tabic types they are lost. They are lost also in psychoses complicated with multiple neuritis, and frequently in cases with diabetes, and in morphinomani. The deep reflexes are exaggerated in senile dementia, many acute affective insanities, hysteria, epilepsy, and in patients with accompanying multiple sclerosis. The state of the superficial reflexes possesses little significance, except in insanity associated with hysteria and organic disorders of the brain, spinal cord, or peripheral nerves.

The Argyll-Robertson pupil is met with almost constantly in general paresis. The pupils in all cases of insanity should be examined as to their equality, size, and reaction to light, and in accommodation. Loss of reaction to light may be observed, besides, in general paresis, in syphilitic insanities, senile insanity, and in some alcoholic cases; it means organic disease of the brain. In rare instances a transitory rigidity of the pupil occurs in epilepsy and morphinomani. Inequality of pupils is very common in organic and occasional in functional insanities.

**Trophic Disorders.**—General disturbances of nutrition, variations in bodily weight, are commonly noted, and possess considerable significance. Thus, rapid increase in weight is characteristic of the progress of an acute psychosis to terminal dementia; if, however, it accompanies an improvement in mental symptoms, it betokens convalescence. In some cases enormous decrease in weight, in association with pernicious anemia, leads to a fatal termination. Certain forms of insanity, especially organic, notably paralytic dementia, present a remarkable trophic disturbance in the bones, a *fragilitas ossium*, inducing easy fracture. Decubitus is observed in bedridden insane patients, particularly paretics.

Hematoma auris, atheroma, or the "insane ear," is a deformity of the ear produced by a hemorrhage into the substance of the auricle, usually between the perichondrium and the cartilage. It is undoubtedly traumatic in its origin, but there is fundamentally some change in the vascular walls in certain cases of chronic insanity, rendering them fragile and easily ruptured by the most trivial pressure or injury. Such effusions of blood do occur in normal individuals (athletes and boxers), but always from severe trauma. The frequency of hematoma auris in general paresis, and in many chronic forms of insanity is only explicable on the hypothesis of some trophic change in the vessel-walls.

**Secretory Disorders.**—The secretion of tears is generally reduced or absent in melancholia.

The saliva may be diminished in quantity in melancholia. More often in many forms of insanity it is increased, the excessive secretion amounting sometimes to a sialorrhoea. The increase is due to constant mastication, to illusions and hallucinations of taste, and sometimes to



irritative stimuli in the secretory centers. Drooling may give the appearance of an increase of salivary flow, because of relaxation of the oral and buccal muscles, or because the secretion is not swallowed.

Diminution or increase of hydrochloric acid in the gastric juice is noted in many cases of insanity, and the quantity may be determined by the Sjögqvist method. Hypochlorhydria exists in common in states of congenital and acquired intellectual defect and in general paresis. Hyperchlorhydria is not infrequently met with in cardiacal attacks, after epileptic seizures, and in catamnestic conditions.

As regards the *urine*, quantitative and qualitative changes are very common in insanity. These changes may be the expression of abnormal metabolism in the central nervous system, of abnormal metabolism in other parts of the body induced by disease of the central nervous system, or of excretory changes in the kidneys brought about by the psychomimetic. Polyuria is observed in many organic psychoses and in hysterical complications. Oliguria is characteristic of melancholy and stuporous conditions. In hysterical insanity there is frequently an alternation between oliguria and polyuria.

As regards the qualitative changes in the urine of the insane, we are year by year recognizing more and more the importance of investigation in this direction. There is no doubt that the deeper our researches go into the chemistry of metabolism and catabolism, the nearer do we attain to a better understanding of the mysterious nutritional processes that have to do with the construction of the blood and that underlie so many psychoses. Albumin, peptone, and propeptone are found not infrequently in the urine of cases of organic insanity, in delirium tremens, in epilepsy, and in acute mania. Their presence is often transitory, and unaccompanied by renal disease. Hyaline cylinders are also often observed in severely excited conditions.

Excessive phosphaturia is noteworthy in many cases of great cerebral excitement, and after epileptiform and apoplectiform seizures. In chronic brain disorders the quantity of phosphoric acid is diminished below the normal.

The chlorids are lessened in quantity in *melancholia*. They are increased in the early stages of *paresis*, but diminish with the progress of the disease to dementia.

Sulphates and the aromatic etheral sulphates (the latter being the product of destructive proteid metabolism) are increased in febrile conditions, and in conditions attended with much tissue-waste.

Urea is also representative of destructive proteid metabolism, and is an index of the general nitrogenous metabolism of the body. It is increased in conditions associated with tissue-waste, diminished in states of malnutrition. Uric acid and the urates have much the same relation.

Oxaluria (any increase above the normal amount excreted in twenty-four hours—viz.,  $\frac{1}{2}$  of a gram) is observed in certain nervous and mental disorders, but its precise significance still requires determination.

Urobilinuria and bilireubinuria have occasionally been noted in general paresis.

Glycosuria, with or without polyuria, has often been observed in various organic psychoses. It may be intermittent, transitory, or permanent.

Acetonuria is encountered in general paresis and epilepsy at times, as also in psychoses attended with malnutrition, as, for instance, melancholia.

Indican should be sought for, as it is an indication of albuminous putrefaction. It is significant of auto-intoxication.

There is a wide region open to the pathological chemist for discoveries in the feces, as well as the urine, of relations between metabolism and psychopathic disorders.

*Menstruation* is often disordered in insanity. Amenorrhœa is the rule in acute psychoses of any form, due undoubtedly to profound changes in the general nervous system influencing the spinal centers for ovulation and menstruation. The cessation of menstruation with the onset of an acute psychosis is often mistakenly supposed by the laity to show some etiological relation between the genital organs and the insanity. The return of the menses is one of the early signs of convalescence from acute mania and acute melancholia. Naturally, it would not be correct to ascribe amenorrhœa in all cases to simply nervous inhibition, because it may arise in all kinds of psychoses as the result of actual genital disease or of marked anemia.

*Temperature-changes in Insanity.*—The physiological oscillations of temperature are greater and more irregular in the insane than in normal individuals. In general, however, insanity may be said to run a non-febrile course.

Subnormal temperatures are frequently observed in melancholia, stuporous states, general paresis, idiocy, and occasionally in conditions of great excitement. In these last they are apt to indicate approaching collapse.

Hypernormal temperatures are found in many psychoses, sometimes from very slight peripheral irritations, such as retention of urine, gastric catarrh, constipation, mild bronchitis, decubitus, sometimes from organic changes in thermogenic centers. Hysterical complications may be associated with hysterical fever. Motor agitation in mania, acute paranoia, melancholia, and so on, may, if marked, give rise to febrile symptoms. The status epilepticus and convulsive seizures of general paresis increase the temperature, as a rule, to a noteworthy degree. Many writers have described diurnal oscillations of temperature, variations from day to day, asymmetrical axillary temperature, and general subnormal and hypernormal conditions of temperature in paralytic dementia; and some years ago, in association with Dr. Langdon, I undertook a verification of these statements at the Hudson River State Hospital for the Insane.<sup>1</sup> These are the conclusions we drew from a study of the temperature in twenty-five cases of general paresis:

1. As regards the average bodily temperature, we find it to correspond in physiological norms. The statements of our predecessors as to hyperpyrexia or subnormal averages can not be sustained.

<sup>1</sup> "A Study of the Temperature in Twenty-five Cases of General Paralysis of the Insane," *Journal of Nervous and Mental Diseases*, Nov., 1902.



2. The diurnal oscillations of temperature in paretics also correspond to physiological norms. The statements to be found in literature as to extraordinary daily variations being frequent in these cases are absolutely erroneous.

3. Asymmetrical axillary differences are so small that they can not be considered as abnormal, and certainly not of any diagnostic significance.

4. When unusual variations of temperature occur in general paretics, their cause must be sought for in conditions not related to the pathological phenomena of paralytic dementia, but depending upon thermogenic features unrecognized by the physician, or "masked" by the mental state of the patient. Thus, in case two of our series, an increasing hyperpyrexia was noted during the second week's observations, but the pneumonia causing it was "masked" until the fifth or sixth day, the patient dying on the sixth day. Again, in case ten, where the highest single daily oscillation was 3.4 degrees, and the average daily oscillation for the week 2.2 degrees, the patient suffered from bed-sores, which undoubtedly produced some septicæmia. That variations of temperature take place in connection with the paralytic and convulsive seizures of these cases we do not gainsay.

**Vascular Disorders.**—The action of the heart and vessels is often influenced by insanity. The pulse is subject to acceleration in excited and neurasthenic states, and to retardation in stuporous conditions. Variations in arterial tension are particularly noticeable at times; arterial spasm in any psychosis, but especially in melancholia, depressed types of general paresis, and in paranoia; arterial paralysis as a sequel to this. No doubt strong mental shocks and depressive or exalting affects are associated with anomalies of the vasomotor innervation. Perhaps many psychoses depend upon cerebral angioneuroses. The apoplecticiform, epilepticiform, and maniacal seizures of general paresis are believed to have their origin in these. Preordial anxiety, the neuropathic cervical globus, and other paresthetic and paralytic sensations in the domain of the vagus, are also, in all likelihood, due to angioneurotic conditions.

## CHAPTER IV.

### METHODS OF EXAMINATION.

THE examination of a patient with mental disorder is a much more complex process than that of a case of physical disease, for it is necessary in the former not only to ascertain the present physical condition, as with ordinary patients, but also to investigate the mental state, which involves the employment of unusual and new methods, and brings us into contact with a novel series of psychic phenomena; and, moreover, to attain our end, we need to study the whole past life of the patient, his diseases, accidents, schooling, occupation, environment, temperament,

and character. Nor can we stop here, for it is of the greatest importance to inform ourselves as to conditions among his antecedents, to determine the type of family from which he sprang, and the presence or absence of an hereditary taint. There is, therefore, much to learn even before seeing the patient in person. The history of a case of insanity, as now recorded in our best insane hospitals, makes a rather formidable volume. It includes every kind of physical record made in general hospitals, as well as a thorough survey of the patient's life and ancestral conditions, and keen psychological analysis of his psychosis and its beginning and progress.

In medicolegal cases we have to guard against several sources of error in our diagnosis, among which are the concealment of delusions by an actually insane patient and the simulation of insanity by a sane criminal. The forms of insanity usually simulated, because of the facility of so doing, are a maniacal state, dementia or stuporous melancholia, and epilepsy with insanity. Recently a notorious individual in New York simulated a paranoïd condition with considerable success. Only one with excellent knowledge of the symptoms of insanity can simulate any form of psychic disorder so well as to defy the skill of the physician familiar with mental diseases.

In general practice it sometimes occurs that peculiar forms of delirium, incident to severe visceral disease, may be at first mistaken for insanity. Thus I have, on a number of occasions, been called upon to assist in the commitment of patients to asylums, where careful examination showed the existence of either a transitory delirium in association with an apoplectic or other organic lesion of the brain, or a delirium from some such visceral condition as Bright's disease. Delirium of this kind is distinguished, first, by the discovery of the associated and causative organic disease, and, secondly, by the usual non-conformity of the delirium to any special type of psychosis.

Were I to formulate a series of rules to guide the examiner in his investigation of the mental condition of a patient, they would be somewhat as follows; yet it is to be remembered that these are not fixed rules, but subject to such modification by the tact, good judgment, and common sense of the examiner:

1. It is to be presumed that previous to seeing the patient the examiner has fully informed himself of all the facts to be furnished by relatives or friends, and has, when possible, inspected letters and other writings, which so often prove fruitful sources of information.

2. Go to the patient as a physician, and not under the pretense of being something else—a device so often suggested by the family and friends.

3. Proceed to the physical examination of the patient, during which tactful questioning will determine the direction to follow in further inquiries.

4. Gain the good will of the patient by kindness and consideration.

5. Even if the patient is distrustful and uncommunicative, be politely persistent, and prolong the first examination, even to the extent of trying the patient, until the object is attained; for many patients



will, when fatigued, finally yield to the friendly insistence of the examiner.

6. If one examination is insufficient, however, have as many interviews as are requisite for the purpose in view—a careful scientific diagnosis. In medicolegal investigations this is especially necessary.

The method of study and investigation then resolves itself into the following:

1. History of the family and of the patient.
2. Observation of the patient.
3. Examination of the patient's physical and mental condition.

**Family History and History of the Patient.**—In the study of the heredity it is often worth while to construct a family tree, showing the relationship and hereditary diseases in the parents, grandparents, collaterals, etc., and it is well to go carefully in every direction among the antecedents, and not be content with too concise a record of this important factor. A special point should be made to determine in each case of mental or nervous disorder discovered whether the disorder present was acquired in the particular individual or in itself probably hereditary. Thus, epilepsy in an antecedent might be acquired (traumatic) and have much less bearing on the descendant than if it were the idiopathic type. Again, general paresis, senile dementia, a toxic psychosis, traumatic insanity, and so on, acquired disorders, as a rule, would have less significance than manic, melancholic, or circular psychoses, as determining factors in the individual case. It should be ascertained whether the parents were blood relations, and whether there was any great difference in their ages. Then the following questions should be answered, not only for each parent, but for the other blood relations, so far as it is possible:

1. Character and temperament.
2. Any special gifts, one-sided talents or peculiar traits, or criminal tendencies?
3. Any insanity? If so, what type?
4. Any nervous diseases, such as epilepsy, ties, constitutional neurasthenia, chronic headaches, migraine, or hysteria?
5. Any constitutional disease (syphilis, tuberculosis, rheumatism, gout, diabetes)?
6. Any alcoholism or drug addiction? Kind, extent, duration?
7. Any congenital infirmity or defect, such as blindness, deafness, dumbness, or deformities?
8. Were there brothers or sisters who died young; if so, of what?

Then a series of questions is given to ascertain the personal history of the patient up until the time of onset of the psychosis:

1. Are there brothers and sisters? If some died young, of what diseases? Any special features in regard to them?
2. Any abnormality in the pregnancy or parturition of mother?
3. Convulsions or other nervous disorders in infancy or childhood?

Date and duration.

4. Rachitis or fibrous diseases in childhood?
5. At what age did patient walk, speak, and complete dentition?

6. Character and temperament in childhood—any precocity, over-  
sided talent or stupidity?

7. Kind and degree of schooling?

8. Period of puberty—was its development normal? Any mastur-  
bation or perversion?

9. What is his occupation, and how has he carried it on?

10. Character, temperament, religion, physical condition, diseases  
during adolescence.

11. Any intemperance in the use of alcohol or drugs? Overwork?  
Shock? Trauma to head? Syphilis?

12. Any evidence of psychopathic constitution?

13. Is the patient married? If any children, are they healthy?

14. Any previous attacks of insanity?

Having obtained the data relating to the family history and the per-  
sonal story of the patient's life up to the onset of the psychosis, we  
ask the following questions in relation to the history of the attack  
itself:

1. Was the onset of the psychosis gradual or sudden?

2. Were there any peculiarities noticed in the patient's conversation  
or behavior, or in his physical appearance, before the insanity became  
apparent?

3. Did he sleep well or badly?

4. Had he lost weight? Did he eat well or little?

5. Has he been excited or depressed or changeable?

6. Has he talked much, little, or not at all?

7. Has he seemed to hear false voices or see imaginary things?

8. Has he seemed to have delusions of suspicion, persecution, or  
of grandeur?

9. Has he threatened or attempted suicide, or violence to others?

10. Any offenses against morals or the law?

11. Any evidence of disorder of memory or defect of intelligence?

**Observation of the Patient.**—Very often the manner of reception  
of the physician by the patient, his facial expression, and bodily atti-  
tude afford strong clues to the type of mental disorder presented. There  
is a vast deal to learn by mere observation without making either a  
physical or mental examination. If it is possible to carry on such  
observation for a time without the patient's knowledge, it will be an  
advantage.

The *physiognomy, attitude, and behavior* will first strike the examiner.  
He should note where the patient is, whether in bed or up and dressed,  
and if properly dressed. A *single melancholic* state is quickly recog-  
nized by the facial expression of depression, silence, and by the hanging  
head, motionless body, or nervous picking at the hair, finger-nails, or  
clothing. In *agitated melancholia* there is an expression of anguish  
and motor activity in the way of restless walking to and fro, wringing  
the hands, tearing the hair, beating the breast, and so on. The *manic*  
patient is extraordinarily lively and cheerful in expression and exuberant  
in speech, gesture, and motion, leaping, running, dancing, singing, and  
talking incessantly.



The *paranoid* expression and manner are threatening, secretive, suspicious, unfriendly, or ironical when the delusions are of a persecutory nature; when grandiose ideas are present, a proud look and majestic bearing are characteristic.

In *demented* conditions, whether late stages of dementia praecox, paresis, or other forms, the face is vacant and imbecile, the attitude completely apathetic, and the gait slipshod and halting.

It is easy to recognize the ecstatic expression of *epileptics* in conditions of ecstasy, when they lie or stand perfectly still, lost in the contemplation of their visions. *Paroxysms* is often recognized at first sight by the unequal innervation of the two sides of the face, the muddy complexion, the overaction of the occipito-frontalis, the unequal pupils, and the frequently coarse tremor of the face when they try to speak.

The *catatonic* type of dementia praecox presents a mask-like, expressionless face, and a completely apathetic pose or rigidity of the body.

The *chronic alcoholic* is often quickly recognized by his swimming reddened eyes, cutis pettoraria, and expression of roguish humor.

This first view, too, differentiates speedily the *idiot* and *imbecile* from other types of mental disorder, and various stigmata of degeneration are frequently at once apparent, serving, by shape of head, character of ears, curious physiognomy, to distinguish the severe types of psychopathic constitution from others.

Negligence in dress, or a tendency to slovenliness and overdecorate, when contrary to the normal habits of the individual, are indicative of a change in the mental state.

If the patient is found in a *stupor*, we may have one of several conditions present, such as stupor with melancholia, hallucinatory stupor of paranoia, catatonic stupor, epileptic stupor, manic stupor, and occasionally stupor in cases of paresis; and the expression of face is often a guide here to a diagnosis of the type of mental disease.

An acute excitement or delirious condition may be due to various states, and one must exclude febrile diseases, alcohol and drug states, and organic diseases of the brain before making a diagnosis of the real psychoses, in which mental and motor excitement occur (such as paresis, epilepsy, manic-depressive states, and catatonia).

*Stereotypy* of attitude, of movement, or of speech may be one of the first symptoms noted in our observation of the patient, and renders the diagnosis of the catatonic form of dementia praecox easy. *Stereotypy* of attitude is a fixed, rigid position, with eyes closed, and every sense unreachably by any stimulus. *Stereotypy* of movement is the constant repetition of the selfsame movement of arms, legs, or face, usually singular or dull in character. *Stereotypy* of speech is the reiteration over and over and over again of words or phrases, often in a peculiar singsong tone. *Stereotypy* is occasionally found in other psychoses, but is most common in catatonia.

*Mannerisms* in speech, dress, actions, peculiar grotesque and clownish or theatrical behavior are also characteristic of the catatonic syndrome. To these we may add the phenomena of *negativism* and *cataplexy*

to complete the picture of this form of dementia *precox*. Negativism is resistance to everything—to dressing, undressing, feeding, to answering questions (nati-m), to passive movements, and the like. The cataplexy may be of the kind produced by automatic obedience or suggestibility. Place the patient in any conceivable attitude, he remains there (waxy flexibility). Set one of his arms going up and down or describing a circle, and he may continue to do this indefinitely (echopraxis), or he may repeat every word you say to him like an echo (echolalia).

Having made these various observations of the patient, if he is talkative and more or less excited, we try to take down, stenographically, if possible, what he says, before asking him any questions or making our physical examination. From his speech we determine whether there is a flight of ideas, what the associations are, whether there are many sound associations, whether it is so rapid as to be "telegraphic speech," whether he makes new words (neologisms), whether the speech is completely incoherent, the presence of reiterations, verbiage; and often from the contents we determine the existence of delusions of a grandiose or persecutory nature and of hallucinations.

From any writing of the patient shown to us before our interview we are frequently able to make a diagnosis. The peculiar elisions and reduplications of letters, syllables, words, and phrases of general paresis are not found in any other mental disorder. The mannerisms in writing, neologisms, and reiterations of dementia *precox* are also characteristic. Furthermore, such letters may reveal the presence of delusions not easily elicited from the patient in conversation.

#### **Examination of the Patient's Physical and Mental Condition.—**

The following points should be investigated as to the general physical condition:

1. General nutrition—atrophy, hypertrophy.
2. Stigmata of degeneration.
3. Skin—old eruptions, scars, cicatrices on penis, mucous patches, bed-sores, swellings, cyanosis.
4. Lungs and heart, pulse, circulation.
5. Blood—pressure, hemoglobin, parasites, differential count.
6. Genito-urinary system, urinalysis.
7. Gastro-intestinal system, stomach and fecal tests if indicated.
8. Osseous system.
9. Abdominal viscera.
10. Sleep.

The examination of the condition of the nervous system should give information on the following points:

1. Reflexes—papillary and tendon.
2. Special cranial nerves—olfactory—optic (hemianopsia, limitations of visual field, color, fundus)—oculomotor (diplopia, nystagmus, proptosis, ptosis)—taste—hearing.
3. Common sensation—anaesthesia, paresthesia, hyperaesthesia, stereognosis.
4. Motor symptoms—paralysis, or morbid movements, gait, ataxia, etc.



5. Speech—any form of dysarthria, or any species of sensory or motor aphasia.

The psychic investigation then begins with a study of the patient's *orientation*. His orientation as regards himself is determined by his answers to the following questions:

1. What is your name?
2. How old are you?
3. What is your occupation?
4. Where do you live?
5. Where were you born?
6. When were you born?

Then his orientation as to time is ascertained by asking:

1. What year is this?
2. What day is this?
3. What is the date and the month?
4. Is this summer or winter?

Orientation as to place is elicited by asking:

1. What place is this you are now in?
2. What city is this?
3. Where is your home?
4. Can you name any of the people about you?

Complete disorientation is observed in some acute psychoses, like some epileptic conditions, acute hallucinosis, and febrile and alcoholic deliria, and also in states of profound dementia. Partial disorientation is found in a variety of other psychoses.

To determine whether the patient has any *insight* into his own disorder we may ask him:

1. Is this your home?
2. Is this a hospital?
3. Why are you in a hospital?
4. Are you sick?
5. Is anything particular the matter with you?
6. Is there anything the matter with your brain or mind?

The *memory* must be tested by all sorts of questions dealing with the whole past life of the patient, questions relating to childhood and youth, the family, the schools attended, the studies carried on, the occupations followed, and so on, in order to determine whether the memory-store for the whole past is intact to date; secondly, whether there are periods of time in which memory-material is missing (certain lapses of memory), and, lastly, whether there is any failure in the power to add to the old store of memories. This last has to do with recent memory material. The Germans call this power to increase the store of memories *Merkfähigkeit*.

Simple tests of memory may be made by having the patient write an autobiographical sketch, by questioning him in relation to school studies, geography, history, religion, literature, by giving him sums in mental arithmetic, by having him repeat old and familiar poems learned at school, and by having him recite the alphabet, Lord's prayer, names of the months, etc.

Defects in memory are often observed early in paresis, and are constant in senile dementia (especially memory for recent events, the old being well preserved), while in Korsakoff's psychosis the loss of the power of adding to the memory-store is a cardinal symptom. Amnesia is particularly characteristic of epilepsy. Fabrication or confabulation, which is a sort of hallucination of memory, the patient trying to fill up gaps in his remembrance by all sorts of remarkable stories, is a symptom found in various kinds of insanity, particularly in some paraneur conditions, in general paresis, in delirium tremens, in Korsakoff's psychosis, and in certain manic and senile cases.

To test the intelligence we have already had the autobiographical sketch, and the answers to questions relating to the whole early life of the patient, but to these tests we must add the following:

1. Have the patient write one or more brief essays on any subject which the examiner thinks him competent to deal with.

2. Write a letter.

3. Draw pictures of objects from memory. Make a map of some country with which he is familiar from memory.

4. Try the word-association method, although this is not as important as a test of intelligence as it is of emotions. Still it has a certain value in showing often a richness of association not found among unintelligent individuals.

*The Word-association Method.*—While the word-association method is of some use for the purpose for which it was first employed, viz., to estimate the intelligence of an individual, it is of even greater value in unearthing emotional complexes. The subconscious is vastly more important to us than the conscious, for in the subconscious lie all the elements that make up our personality, not only the treasury of all our individual experiences through the course of years, but all our ancestral trends, desires, tendencies, wills, ambitions, controls, inhibitions, fears, in fact, the latent spirit of the race of mankind.

Each man's vocabulary, be it the three hundred words of the sailor or the fifteen thousand words of a Shakespeare, or the average fifteen hundred or two thousand words belonging to us, is related to all that subconscious material. A word has a magic power in it to summon from the vaults of memory all sorts of apparitions. Each word has an emotional value, some more than others, because all of our deepest experiences are associated with the words we know.

So when an apparently empty word is propounded to a patient, and he is asked to answer with another word as quickly as possible with the first word that comes into his mind, we not only obtain an association from his memory storehouse, but we may strike some emotional complex which is indicated by a slow response to the test word or retarded reaction time.

This is the word-association method of Jung, a method that is employed for the discovery of secrets in the criminal, or painful and disease-producing emotional complexes in patients suffering from various psychogenic disorders. In making this test just ordinary, everyday words are used, since these are the special words related to an ordinary



individual's experiences, and a fifth of a second stopwatch is used to measure the reaction time. An emotional complex is so apt to have many words associated with it that there is an influx of many words to the stimulus word, and the mind panics for a choice; hence the retarded reaction time. Having gone over the list once with a stopwatch, we go over the same list of words again to see how well the first associations are remembered. The influx of words is responsible for faulty memory here, and where there are emotional complexes, these reproductions are apt to be false, some new word being associated the second time.

Thus, a patient was given a series of unimportant words that had no significance whatever to the investigator, and among them the following had three or four times as long reaction time as the others, so that it was clear that an emotional complex lay behind them: Water—deep, 5 seconds; ship—sink, 3.4 seconds; lake—water, 4 seconds; swim—can swim, 3.8 seconds.

Psychoanalysis showed that the patient had recently been depressed, and had determined to commit suicide by drowning.

In making up our list of words, we select the most common words in everyday use, and propound them in series of twenty-five to fifty or a hundred. It is well to vary the character of the words in the list by writing down a noun, an adjective, a noun, a verb, and so on, rather than to make the series all of one grammatical genus. Here and there in such lists words thought to have special significance to the patient may be introduced.

*The Galvanometer as a Measure of Emotions.*—We may employ a Deprez-d'Arsonval galvanometer at the same time with the word-association test, and the fluctuations of the galvanometer in proportion to the extent and reality of the emotions is important in corroborating the evidence given by retarded reaction time and false reproductions. One or two cells are placed in the circuit with the galvanometer and the patient, who has his hands upon metallic plates. Emotions stimulate the sweat-secretory glands, and by thus reducing resistance in the circuit, determine fluctuations of the galvanometer mirror. A light thrown upon the mirror is reflected upon a measured screen concealed from the patient, and the extent of deviation may be accurately estimated and set down. The whole procedure and the results in normal and insane persons were fully described in *Brain*, August, 1907, by Jung and myself.

*Psychoanalysis.*—This method of psychological study of a patient, developed by Freud, is more often applicable to cases of hysteria and psychasthenia than to insanity, but it is frequently of use in minor psychoses. Freud's technique is as follows:

The patient lies quietly on a sofa in order to be tranquil and to avoid physical or other distraction. The physician sits at his head, and may at times place his hand upon the patient's forehead, a physical stimulus which often assists the patient in concentrating his attention upon the subject in mind. This may be and often is some emotional complex buried too deeply in the mind to rise readily into consciousness, some emotional experience having a causal relation to the psychoneurosis.

Perhaps the word-association test has already been used, and clues thus afforded for this additional investigation. The patient is urged to talk freely and frankly about his symptoms and their origin. Often the memory is found to be at fault, and he is requested to tell everything that passes or comes into his mind, even if painful and embarrassing. We may utter some significant word known to be a clue, and then ask him what thoughts occur to him in connection with the words. We may also ask him to repeat to us his dreams, and from these, which have their being in the subconscious, we also draw material for our purpose. The object of such psychanalysis is twofold. In the first place we may uncover the details of some psychic trauma which is usually at the basis of most hysterical manifestations and of many psychoneurotic conditions, such as obsessions, phobias, and the like, and may be the origin of some type of insanity, like *dementia precox*. In the second place, it is a therapeutic procedure, and the psychanalysis ordinarily cures the patient. There is a psychological mechanism associated with all painful, unendurable emotions. Either the emotion is reacted to adequately at the time, as in normal grief, or it is intentionally suppressed. The intentional suppression is helped by the wish and struggle to forget. If one receives an insult and knocks the giver down, this is an example of an adequate and satisfactory reaction to a disagreeable emotion. If one is obliged to "pocket the insult," a very good expression for the mechanism in question, it will fester indefinitely, perhaps for years, in one's bosom, suppressed, possibly forgotten, but liable at any time to light up by sudden association of place or face or word. Suppose a child is assaulted, a young woman jilted on the eve of marriage, a young man overhears some jeering remark concerning himself—these are psychic traumas which, without adequate reaction, may become suppressed emotional complexes acting like a parasitic body on the psychic, drawing all sorts of associations to itself, and finally, by a process of conversion, may react upon the physical organism, inducing hysterical pains, anesthesias, palsies, and the like. If by psychanalysis we are able to reawaken the old painful memories and discuss them with full circumstance and detail, with free play of the emotions, the hysterical and other psychoneurotic symptoms disappear. This *abreaction*, or reacting off, has for ages been taken advantage of by the confessional of the Catholic Church, those wise fathers long ago recognizing the psychological fact that a secret remorse, pain, or grief, unburdened to another, lifts the load from the penitent and suffering.

*The Study of Patients' Dreams.*—It was Schopenhauer who said that insanity is a long dream and a dream brief insanity. There is, in fact, more than a superficial resemblance between dreams and insanity—so much so that psychiatrists the world over are devoting themselves to the study of dreams as a part of their clinical and scientific work. There is practically no phenomenon that presents itself in dreams that we may not observe among the inmates of an asylum ward. There are in both mental disease and dreams a prominence of visual and auditory hallucinations, a tendency to the reproduction of old experiences, the imaginary fulfillment of wishes and desires, bizarre associations, chaotic



flight of ideas, incoherence, disorientation, weakened judgment, and division of personality.

Sometimes insanity first manifests itself in dreams, though the mind is still normal by day. In alcoholism dreams sometimes foreshadow the characteristic alcoholic delusions (of infidelity, etc.), and dreams may be the equivalents of epileptic seizures. Sometimes in patients just recovered from insanity who are normal by day there is a nightly recurrence of insane delirium in sleep—a species of nocturnal insanity. A terrible dream may usher in insanity, which then concerns itself with the material created by the dream. Dreams at times induce the imperative ideas and impulses of psychasthenia. Thus, in a way, we may look upon insanity, in itself a pathological condition, as a summation of periodically recurring normal dreams, as a kind of reduction of consciousness, such as exists in the dream state.

Freud has written a fascinating book on the *Interpretation of Dreams*. I can only describe briefly what his views are. In the first place, dreams always seem to be the fulfilment of some wish or desire. In children this is invariably true, as will be found by a little investigation. The child receives in dreams the delights that he longs for. When a dream is recalled and examined, we have before us a curious piece of conglomerate, often uniting materials from the days of childhood with experiences of yesterday, full of absurd distortions of events and words, transformations, allegories, and symbols. There is nothing accidental in the arrangement, and by psychoanalysis of the person having the dream, one determines the origin of every patch in the embroidery; and by investigation one discovers the wish at the foundation which is directly or indirectly fulfilled by the dream. Freud has been much criticised because in his opinion it is usually a sexual idea which is the *force et origine* of these dreams, as well as of hysteria and other psychoneuroses. Doubtless he has gone to extremes in this idea, but he has certainly opened up a very important field and discovered new methods of approach in the domain of morbid psychology.

There are also methods of studying the attention, apprehension, apprehension, and morals, and for eliciting information as to the presence of illusions, hallucinations, and delusions, but in the conduct of an examination of a mental case, as described in this chapter, much will present itself to throw light on those other qualities and symptoms.

## CHAPTER V.

## GENERAL TREATMENT OF INSANITY.

It is not so long a time since the insane in Christendom were believed to be possessed of devils and accursed. On the other hand, in certain parts of heathendom (among the Mohammedans) it was supposed that the souls of the insane had been removed early by God as a special mark of favor, and that they were, therefore, blessed. Medieval treatment was founded upon the curious pathology just described. One portion of the world ducked, whipped, tortured, chained in dungeons, and occasionally burned, the insane. The heathen treated their insane, upon the whole, comparatively well.

After a time, many of the therapeutic measures employed by the Europeans of the middle ages were abandoned as unsatisfactory. But society still had to be protected; so the insane were fettered in the cells of jails and fortresses and solitary towers, until a realizing sense of the inhumanity of such treatment struck a responsive chord somewhere in the breast of a Tuke, a Connelly, a Pinel, a Rush, a Kirkbride, an Earle, and doubtless other, but unknown, immortals both before and after them.

Insanity thus gradually came to be looked upon as a disease, and not a penal offense, and, instead of prisons, special buildings were set apart for the particular custody of the insane. The great object of the asylums at first was to afford protection to society from lunatics, to protect them from themselves, and to provide for their care and support, when at public cost, in an economical manner. A hundred years ago, however, the asylum was still a species of jail, for its evolution had not yet proceeded far. Dungeons and iron chains and staples in stone walls and stone floors were still in use in many places. Indeed, it is scarcely over eighty years since Norris, a patient in Bethlem (Bethlehem Hospital), in the great Christian city of London, was kept for twelve years in a cell, with an iron collar riveted around his neck and iron bands and rings around his wrists, arms, and ankles, the neck being fastened to the wall and the leg to a rude box of filthy straw.

Asylums have, at the present time, come to be recognized as hospitals, and they are approaching nearer to that ideal every year. Occasionally, one finds among them some rudimentary appendage which is reminiscent of the embryonal stage of their evolution; but this is, fortunately, rare. The well-conducted hospital for the insane, to-day, is different from the asylum of years ago: the depressing, barren halls and wards and naked floors have given place to pleasantly furnished and carpeted, cheerful-looking parlors, sitting-rooms, and bed-rooms; muffs and strait-jackets have disappeared; the unintelligent attendant has, in many instances, given place to the trained nurse; every new



means of treatment is carried out to the best of the ability of the asylum physicians; schools, employment, theatricals, music, and out-of-door walks are provided in the place of the old, deadly monotony, and, in fact, the asylum has gradually undergone a metamorphosis, until its character has completely changed. There are, to be sure, not many perfectly ideal institutions as yet in existence, but there are some which approach very nearly to it, as, for instance, that at Alt-Scherbitz, near Leipzig, and the new asylum at Rome, both of which I visited and described in 1887.<sup>1</sup> These are, of course, constructed on the cottage and pavilion plan, so arranged as to impress one as small colonies or villages, with separate buildings for those merely there for custody because of dangerous propensities, those brought there to be cured for kindly during the remainder of their useless lives, those who carry on various occupations, and, finally, for such as enter particularly to secure treatment for the brain-malady which has bereft them temporarily of their reason. The colony system of caring for the dependent classes—which the writer thinks should ultimately be adopted for all kinds of defectives—is well exemplified by the Craig Colony for epileptics in the State of New York. Some day we shall come to realize in all our hospitals and retreats for the insane, it is to be hoped, the ideals already exemplified in two unique institutions for the insane, which are not as well known as they deserve to be. One is the *Maison de Folie*, at Vincennes, in the outskirts of Paris, established nearly one hundred years ago by a famous alienist, the other, singularly enough, is at Iwakura, Japan, and was founded almost a thousand years ago, with a history and evolution similar to that of Ghent, in Belgium. I have described both of these institutions in a paper read in 1910 before the National Conference of Charities, published in the transactions for that year.

I will say that I believe improvement and reform are constantly going on in asylums throughout the world; that no one is more anxious than are their superintendents to make progress in the care and management of the insane. They are rapidly reaching the best methods of dealing with the insane poor. If any are tardy in this advance, it is because they are so often hampered by the never-ending overcrowding of our public asylums, by the interference of politics, by the lack of money, by the want of a sufficient number of medical assistants, and by a multiplicity of official duties.

While these statements are undoubtedly true,—and great credit is due the asylum physicians of the present day for their strenuous efforts in behalf of their charges,—I believe that the ideal treatment of almost any insane person is to be sought outside of an asylum. After an asylum experience of some years, and an experience of many years, too, in private practice, I feel that I am in a position to judge fairly well of the relative merits of treatment in and out of asylums.

Theoretically, it ought to be the right of every individual in sickness to receive the best treatment that medical science affords; but this right can be enjoyed by very few. There are too many interfering conditions. Not every injured man is within reach of the best surgeon; not

<sup>1</sup> "Some European Asylums," *Am. Jour. Insanity*, July, 1887.

every fever-stricken one convenient to the best physician; and few are the deaf, the blind, the lame, those with crippled bodies and those with disordered minds, who ever really receive the best treatment that the world can give. The intelligent doctor and the scientific skill are not the only requisites. Other conditions are good nursing, the most suitable climate, the best hygienic surroundings, the best moral atmosphere. In dealing with affections of the body solely, there is often much to be desired; but it is particularly in the treatment of those who are mentally as well as physically afflicted that so much which should be done is left undone. The obstacles in the way of securing the best treatment are multiplied in the case of the insane by the derangement of the supreme centers of psychic function.

Just as a hospital is a better place than a tenement house for a surgical patient or a case of fever, so is the asylum superior to the home in the care-taking of the pauper and indigent lunatic. The neurotic insane of the poorer classes are best treated, at present, in our large public institutions; and those among the moderately well-to-do, either at home or in the small private asylums. Only the insane of the wealthy classes can, perhaps, enjoy and carry out ideal methods of treatment in their own homes, in country houses, or in foreign travel.

It is, of course, needless to say that there are many degrees of insanity; that there are hundreds of cases that are never obliged to go to an asylum at all; that in society are many insane persons carrying on legitimate occupations and caring for themselves and families; and that, on the other hand, there are cases for which nothing but commitment to an asylum would be suitable or feasible. But we should not send any patient to an asylum unless he needs restraint because of danger to himself or others, or because proper treatment and supervision are difficult in his home, owing generally to poverty or other insurmountable conditions. The sooner a case of acute insanity occurring in a pauper or an indigent is removed to an asylum, the better are his chances for recovery. This merely signifies that the earlier treatment is undertaken by those who are familiar with the management and care of the insane, the better for the patient. Early treatment by physicians of experience in psychiatry is demanded. At present this end is best attained by resort to the asylums of the neighborhood. But the writer has often called attention to the need of increasing and extending the facilities for the early treatment of the insane—a matter which can be accomplished in several ways. The lines of progress in such direction are:

(1) The opening of special reception-wards or pavilions for the insane in general hospitals; (2) the establishment of psychopathic hospitals in large cities; (3) the creation of outdoor departments in connection with asylums situated in densely populated districts.

Before taking up the matter of the treatment of insanity, a few words should be said as regards

**Prophylaxis.**—Naturally, the question of the proper care and education of children with a tainted line or lines of ancestry often comes before the physician. Much can be done to ward off impending future evils by due and early attention to the mental and physical evolution



of such children. One can not begin too soon to regulate the life of these little ones. The very milk of a weak and anemic mother may diminish the feeble resistance of a degenerate child. From the day of birth the prophylaxis must begin. The points to be observed in the effort to accomplish this are as follows:

1. Cultivate the body of the growing child. Develop him physically by careful and regular diet, regular hours of sleep, outdoor life, efficient systems of exercise.

2. Let his training be muscular rather than intellectual, manual training rather than lessons, especially in the early years of childhood. No schools until the age of seven or eight years.

3. The child with degenerate tendencies should be forbidden all nervous stimulants, such as tea, coffee, wines, beer, tobacco.

4. Seek to develop the resistance of the organism to all external stimuli, hardening his body by the daily morning cold bath, frictions, exercise, a hard bed, a cool sleeping-room; accustoming his mind to the courageous endurance of pain and mental stresses.

5. Guard well the epoch of puberty.

6. Let the occupation chosen for later years be also one for the muscles rather than for the mind, an outdoor rather than an indoor calling, a country rather than a city life.

Many of our States and many other countries are taking up very seriously the question of the prevention of insanity, either through various charitable bodies or through the activities of societies first founded for after-care of the insane. The after-care societies have broadened their field of work in many cases by adding fore-care, or prevention, to their functions. It would seem as if more than 40 per cent. of insanity could be prevented by the eradication of alcohol and syphilis alone as causes. By the crusades and public education now being carried on everywhere, there is no doubt that there will be a gradual diminution in insanity due to these causes. It is possible that the discovery by Ehrlich of salvarsan may lead to a more certain means of destroying the germs of syphilis, and, while as an aid in the treatment of paresis it is apparently of no use, as a means of prevention of paresis it may prove successful.

**Isolation.**—On being called to see a patient suffering from insanity, the first point which arises is whether he should be sent to an asylum or not. This is generally a question of means. Isolation from the immediate friends is in nearly every case a requisite. If the patient belongs to the indigent or to the middle classes, isolation and the best treatment for his ailment are only to be satisfactorily obtained in an asylum or hospital for the insane. Among the well-to-do, the needed isolation may be successfully secured in his own house, in an ordinary sanatorium, or by means of travel with a suitable nurse, companion, or physician. The kind of treatment best adapted to the nature of the case must be decided by the physician. The quiet of a private house in the city or country is best for some cases, while the tonic and stimulus of foreign travel are indicated in others. It may be stated that, when travel seems to be the prescription required, the greater the change from the envi-

resort in which the mental disorder developed, the better. The cities of Great Britain and the Continent do not differ essentially from our own cities, and patients should not be sent to such places with the idea of securing a change of environment. Norway in summer, Egypt in winter, and Mexico in either summer or winter, are regions which offer the greatest inducements in the way of tonics to the nervous system and stimulus to the mind, and all three are, at the same time, peculiarly restful and calnitive.

If these methods of home, country house, or travel are for any reason impracticable, then the smallest private asylum that can be found is to be selected, for the fewer other insane persons and the greater number of sane persons the patient comes in contact with, the better will be his chances for recovery. There is a need for physicians in practice in the country who will be duly authorized and empowered by law to receive in their own homes and care for one such patient. The chief drawback in home-treatment, if long continued, is usually the bad effect of association with an insane person upon other members of his family, particularly if they be neuropathic. With a sufficiency of nurses and room, there is no contingency in the treatment of the insane that can not be guarded against. These being provided, the worst features in a case, such as violence, homicidal and suicidal tendencies, attempts at self-mutilation, etc., may be as well avoided outside as inside of an asylum. There are cases in which—though I am opposed to mechanical restraint in great measure—I should employ long-sleeved night-gowns, or even camisoles, rather than let them go from home before all means of cure had been tried at least for a few weeks' time.

The conditions and propensities that we have to combat are many. The choice of method must be the result of careful deliberation, and after judicial survey of all the features presented. We usually need the assistance of skilled and experienced nurses. Thanks to the asylum training-schools, there are numbers of such trained nurses of both sexes to be had in our large cities.

**Psychotherapy.**—Isolation and psychotherapy may be set down as the great cardinal principles in the treatment of the insane. Under the heading of psychotherapy are included, not only the influence of sane companionship with the best nurses obtainable, and the conversations with them and the medical man in charge of the case, but all sorts of exercises and occupations. One must remember that the cases of acute insanity requiring bed treatment are in reality rare. The vast majority of the cases are rather insidious, insidious, chronic in their onset and course. It is an axiom with the psychiatrist that nothing is better in the way of treatment for the disordered mind than physical employment, which improves nutrition, acts as a safety-valve for the escape of surplus energy, distracts the attention and engages the interest of the patient. Unfortunately this system of psychotherapy is seldom well carried out anywhere in public or private asylums. In public hospitals its inadequacy is due chiefly to lack of money to furnish the necessary teachers of industries, arts, and crafts. In private retreats, the money is not lacking, but the



disposition to use it is absent. The habit of keeping only a boarding-house for the insane is a hard one to break. It is probably true that the doctors in charge of private retreats are not wholly responsible for the fact that they do little for their patients beyond feeding, housing, and guarding them. The defect is partly due to long-established custom, partly to therapeutic pessimism, and partly to the stand taken by patients' relatives who are not yet educated to the occupation-idea. In the ideal sanitarium for mental cases, the indoor and outdoor sports and exercises should be developed to the farthest possible point, under the supervision of a competent director, while teachers of arts and crafts should preside over the handicraft shop, and other instructors should be employed for industries of various kinds, such as agriculture, carpentry, floriculture, gardening, and the like. The system has been well developed at the Craig Colony for Epileptics. Among the arts and crafts that should be taught in every institution for the insane are wood-carving, leather-work, tapestry-weaving, basket-work, pottery, brass-work, drawing, designing, painting, and outdoor photography. Every alienist with imagination knows how very far we are as yet from an ideal standard of care of the insane. But we have corroborative evidence from the side of the patients themselves, as, for instance, in "The Mind that Found Itself," by Clifford W. Beers, who was a patient in several institutions for the insane, both public and private (Longmans, Green & Co., 1908), a book which every "friend of the infirm of mind" should carefully read. See also page 787 on moral treatment.

### TREATMENT OF ACUTE CASES.

In acute cases, whether of mania or melancholia, it has been my experience that confinement to bed is a valuable factor in cure. Hence, on being called to such a case, I have the patient put to bed. Due precautions are taken as to the removal of all sharp instruments, weapons, drugs, cords, door-locks, and the like, and by a simple device the windows are arranged that they may not be opened beyond six inches; otherwise the furnishings may be left as they are without attention.

Insomnia and mental and motor excitement most frequently demand our best skill. In emergency, I am in the habit of using duboisin sulphate hypodermatically in the dose of  $\frac{1}{16}$  of a grain, or sometimes lysocyanin, or lysocin hydrobromate in doses of from  $\frac{1}{16}$  to  $\frac{1}{8}$  of a grain hypodermatically, though these latter are not so satisfactory as duboisin. But for routine treatment of insomnia and mental excitement I much prefer hydrotherapy to drugs. In some cases the prolonged warm bath (70°-80° F.) for from one-half to two hours may be used, but in all cases the hot wet-pack is applicable. Sometimes when the wet-pack does not suffice to quiet fierce mental excitement, I use duboisin in addition, or give doses by the mouth of paraldehyde, trional, and sulphonal, all of which are valuable hypnotics.

In acute depressed conditions, on the other hand, spirits usually act

best in cases in which hydrotherapy does not subdue the insomnia, distress of mind, and disordered nervous system. Among opiates, codein seems to offer advantages over others, and the contraction of a habit need not be feared. The aqueous extract of opium or morphin may be given hypodermatically.

The refusal of food is another element of danger. Acute insanity, besides rest in bed, quiet, and repose, needs overfeeding to balance the great waste of tissue going on in the system. While many cases of acute mania will eat and drink ravenously at times, from the nature of things their actions are uncertain, and the nurse should be instructed to feed the patient almost hourly and keep account of what is given. Milk, raw eggs, meat-juice, and occasional stimulants must, in extreme cases, be our chief reliance. Having an intelligent and assiduous nurse at hand, the necessity of feeding with a tube will only rarely occur. When required, the soft rubber stomach-tube may be introduced by the physician through the mouth or nose, a funnel attached, and the liquid mixture of the substances named allowed to flow in.

There are cases (some of the insanities of puberty and adolescence, and other forms) in which amphetrodines modify distinctly the trend of delusions. There are cases in which intestinal antiseptics achieve noteworthy results; indeed, the instances are few in which attention to morbid states of the alimentary canal is not rewarded by considerable benefit to the mental condition of the patient. Argument with patients upon delusions, more or less fixed in character, often has, despite the opinions of numerous alienists to the contrary, decided value in altering their beliefs, and at times even eradicating their insane ideas altogether. It is true that occasional argument is generally of no avail. Such moral treatment must be solidly and perseveringly employed, daily and for weeks or months, to insure success. Argument is a species of suggestion. The tactful and judicious physician will not make use of it in cases where it leads to irritation and would seem to be injurious.

The most important remedial agents employed in insanity are as follows:

**The Rest-cure.**—This has already been briefly referred to. It was in 1860 that Hilton began his series of lectures on rest and pain, in which he pointed out how much rest had to do with growth and repair of the bodily tissues, and fifteen years later Mitchell wrote of the value of rest in the treatment of hysteria and neurasthenia. Nowadays, however, we apply the principle of rest to a great variety of nervous disorders. Besides its indication in many cases of hysteria and neurasthenia, we find it of the greatest benefit in all sorts of nervous and mental troubles, and especially in such as evince a tendency to waste of tissue and to exhaustion.

Most cases of acute mania need to be treated by rest, which should be made as absolute as possible. Many cases of acute melancholia recover more quickly when confined to bed. While in many mental cases the rest should be absolute for a period of several weeks in order to insure a successful termination, it is astonishing how much benefit can be obtained by a modified rest treatment—that is, by merely pro-



longing the daily amount of repose in bed. The principle is to apply rest methodically, and in proportion to the degree of nervous exhaustion, strain, or irritation.

When rest is made nearly absolute, it is necessary that tissue metabolism should be encouraged by attention to the amount and quality of food, and especially by substitution of some passive artificial exercise for the active movements upon which the organism has hitherto depended. This is accomplished chiefly by massage.

**Massage.**—Massage was a favorite remedy and luxury in ancient Roman times, when it figured as the *Aliptic Art*;<sup>1</sup> so then it is not at all a new remedy, but its vogue in recent years has assumed enormous proportions, and it has received a scientific study and systematization to which the ancients were strangers. This rubbing, beating, and kneading of the trunk and limbs, when skilfully done, is an essential adjunct to the absolute rest treatment. It is invaluable in many kinds of pain, and it often surpasses drugs as a soother of irritation and an inducer of sleep.

**Diet.**—It is needless to say that in connection with a form of rest treatment simplicity should be the rule as regards food. The selection should be made from the point of view of easy digestibility, and foremost in this regard stand milk and its various preparations. Where milk can not be taken in its ordinary form, some more digestible preparation may be employed, such as peptonized milk, koumiss, mutton, or soured. In cases undergoing a rest treatment this is the main staple of food, and it should be given frequently and in considerable quantity. Overfeeding is indeed another principle in the treatment of any of the nervous and mental diseases in which exhaustion is a feature. Thus, absolute rest and overfeeding must be our chief reliance in acute mania, and in severe types of melancholia. Many cases require feeding every hour or two hours. Raw or soft-boiled eggs, rare or rare beef, specially prepared cereals, and sometimes green vegetables and fruits may be added to the diet. (By specially prepared cereals I mean simple boiled rice, stale bread in the form of toast, or, better, bread which has been twice baked—Zwieback). Stimulants are only occasionally indicated, and then especially in acute maniacal or other dangerously exhausting conditions.

A somewhat similar form of diet is appropriate for mental disturbances having a rheumatic or gouty diathesis as a basis. The same diet is essential in all cases of insanity, neurasthenia, epilepsy, and so on, which seem to depend upon auto-intoxication from fermentative or putrefactive changes in the intestinal contents, and such cases we find nowadays to be not at all infrequent.

**Hydrotherapy.**—When in 1893 I wrote a paper on "Hydrotherapy in the Treatment of Nervous and Mental Diseases" (*Amer. Jour. of the Med. Sciences*, February, 1893), there was really no place in the city of New York to which one could send patients and have his own ideas as to treatment faithfully carried out; nor did I know of a single

<sup>1</sup>—*The Aliestic Art: a Historical Study*, by the author, *Phil. Med. News*, Aug. 11, 1893.

asylum for the insane in this country installed with hydrotherapeutic apparatus, such as I had seen in a number of asylums abroad, even in so remote a country as Greece. Now I could name many public and private asylums which are equipped with arrangements for this purpose.

Water affects the nervous system in a variety of ways.

Cold baths increase and warm baths diminish the irritability of the brain and spinal cord in a reflex manner by stimulating the sensory and motor nerves of the skin, thus influencing the cerebrospinal circulation.

For some years they have been using in German asylums the continuous bath for cases of excitement. The patients stay day and night in these baths, sometimes continuously for weeks or even months. Occasionally the continuous bath is used only five, six, or eight hours per day. The water is kept at a comfortable tepid temperature. No very serious skin disorders are produced by such prolonged immersion, and the method is well adapted to cases of excitement.

Short cold baths, especially when combined with sprinkling, showering, or rubbing, are powerfully stimulating, exhilarating, and tonic. Cold baths stimulate peristalsis and the visceral reflexes in the cord, and increase blood-pressure. Prolonged warm baths, steam and hot-air baths, and the hot pack are relaxing, fatiguing, and tend to induce sleep. Warm baths diminish arterial tension and reduce the irritability of individual nerves and the whole nervous system. The spinal douche is of the greatest service in many nervous disorders, because of its remarkable tonic, revulsive, and derivative effects. It is a powerful mental as well as physical stimulus. By means of various nozzles it is ejected in the form of a strong stream up and down the back of the patient for a few seconds only, at a distance of some ten feet. Patients with good reaction do not need any special preparation, but at the beginning it is well to have the patient take a warm bath or stay a few minutes in a hot-air box previous to its application. At the first showers the water should not be too cold. Later, it may be gradually lowered to 50° F. It should be taken every day, when possible. Occasionally this cold spinal douche is alternated with a hot douche (the so-called Scotch douche). This is an exceedingly successful procedure in many cases of hysteria, neurasthenia, and in lethargic and hysterical forms of insanity, where there are sluggish intellect, great depression, apathy, stupor, cataplexy, etc., and in any case of nervous and mental disease where anemia, chlorosis, or gastric trouble exists.

In insomnia there is no other remedy so generally efficient and at the same time so innocuous. I have seen it successful in wakefulness from every kind of cause, and in cases seemingly intractable to other remedies. There are two hydropathic procedures for the production of sleep. One is the prolonged warm whole bath, at a temperature of 70° to 90° F., for from one-half to two hours just before retiring. This is indicated in mild cases of insomnia. But the hot wet-pack is more effectual and more widely applicable in all forms of sleeplessness, whether in nervous or insane individuals. It is applied



in this way : A blanket, nine by nine feet, is spread upon the patient's bed, and upon this a sheet, wrung out dry after dipping in hot water, is laid. The patient lies down upon this, and the sheet is at once evenly arranged about and pressed around the whole body, with the exception of the head, after which the blanket is also immediately likewise closely adjusted to every part of the patient's body. Other dry blankets may now be added as seems necessary. The patient remains in this an hour or longer ; all night, if asleep.

I know of no better treatment of acute maniacal conditions, for instance, than rest in bed, overfeeding, the hot wet-pack, and the occasional employment of some sleep-producing agent.

**Treatment of Auto-intoxication.**—Researches in the physiological chemistry of digestion, as well as observations in many pathological conditions, have established that auto-intoxication from the absorption of poisonous substances generated in the alimentary canal by putrefactive and fermentative processes, or in the various tissues of the body by a perverted chemistry, is not only a real thing, but a frequent factor in the etiology of a number of nervous disorders, such as headache, neurasthenia, hysteria, neuralgia, and even graver maladies, like epilepsy, melancholia, mania. It behoves us, therefore, in these diseases, to investigate carefully for evidence of any such cause. Periodical or constant attacks of gaseous diarrhoea are somewhat indicative of this condition. Frequently the condition of the bowels furnishes no information of the actual state of affairs. Recent researches tend to show that an excess of ethereal sulphates in the urine (malicum) in connection with other symptoms is a good index of auto-intoxication.

When auto-intoxication is suspected as the causative factor in any nervous disorder, it is essential to regulate the diet in the manner already mentioned, and there are at our disposition a number of intestinal antiseptics which, though not always efficient, are yet often of very great benefit. I have found, in my own practice, that bismuthol is one of the best intestinal antiseptics. I give it in capsules of five grains each, two hours after eating, with water. In several cases of epilepsy and of melancholia it has acted exceedingly well. In many cases of epilepsy salicylate of soda has also proved itself of great value. Salol, too, is a good intestinal antiseptic. Sometimes I have made excellent use of peppermint for the same purpose. I think the abundant use of water a necessary adjunct in the treatment, usually advising the drinking of hot water several times daily on an empty stomach, and sometimes adding thereto frequent flushing of the large intestine with warm water.

**Electrotherapy.**—General faradization with a current sufficiently strong to contract the muscles has much the same value as massage where the rest-cure is employed; it exercises the muscles and stimulates metabolism. Over and above this it has a tonic effect. Galvanism is only of use in complicating conditions, such as neuralgias, sciatica, and the like. The same is true of the static and sinusoidal currents. Electrization of the head for the purpose of influencing illusions, hallucinations, and delusions is occasionally of service, but doubtless its

influence is almost wholly of a suggestive nature. However, it is not to be interdicted on that account, for suggestion is in itself a valuable therapeutical adjunct, and so good a method of increasing its usefulness as is afforded by electricity is not to be slighted. Suggestion is a species of psychic therapy.

**Drugs.**—The narcotics are of great importance in the treatment of insanity. Among these, opium and its alkaloids easily stand first.

**Opium, morphin, codein,** all have a hypostic effect, but their especial value lies in their sedative influence upon mental hyperesthesia, anxious states, etc.; in their contraction of the blood-vessels, and in their stimulation of the nutrition of the central nervous system. The hypodermatic use is best. They are particularly indicated in melancholia, acute alcoholic psychoses, and hallucinatory paranoia, very seldom in maniacal states. They are contraindicated in most maniacal conditions, collapse, fatty heart, uncompensated valvular disease, and marasmus. The patient should not know the name of the drug used. Opium and codein are preferable always to morphin, because of less danger of forming a habit. The doses must be gradually increased. The constipation at first present during the administration of opiates disappears later.

**Hyoscin, hyoscyamin, and daboisin** are isomeric alkaloids, and have much the same qualities and are alike in their effects upon the organism. Next to the opiates they form the chief drugs of the alienist's armamentarium. Their great value lies in their sedative influence upon motor centers. They are used hypodermatically in doses of from  $\frac{1}{16}$  to  $\frac{1}{8}$  of a grain. Almost immediately after injection the muscles become incoordinated and weak, and in ten or fifteen minutes the patient sinks into a light slumber which lasts from six to eight hours. The peripheral arteries are contracted, giving the patient a striking pallor; the breathing is slowed, the pulse retarded or made intermittent, the throat rendered very dry, and the pupils enlarged and accommodation paralyzed. These drugs are contraindicated in heart disease, and in no case should they be continued any length of time. Perilous as they are on the right occasion, their employment should be subject always to the careful and judicious supervision of the physician.

Another feature of their physiological action to be borne in mind is their power to induce dreadful hallucinations in a well person—a fact which emphasizes the need of care in administering them to an individual whose mind is trembling in the balance. Long-continued use of these alkaloids interferes with nutrition.

From what has been said of the action of these drugs, it will be seen that their effectiveness is most manifest in conditions of motor excitement, in mania, agitated melancholia (combined with morphin), in agitated dementia, and in the motor excitement of epilepsy or paresis. I have often been able to feed excited patients who refused food, immediately after the injection of the alkaloid, during the few minutes that elapse before the advent of sleep.

The bromids, aside from their particular value in epileptic psychoses, are often useful in other forms of mental disease, owing to their



effect in diminishing cerebral activity and reflex irritability. In epileptic insanities the combination of the bromids and opium is especially effective. They are of use in any mental excitement which is combined with some reflex irritability (illusions and organic sensations, uterine and genital disorders). As an anti-aphrodisiac they are employed in insanity with erotic manifestations. In large doses, sixty to ninety grains and over, they act well as a safe and innocuous hypnotic.

**Chloral hydrate** is not so much used as formerly, though its hypnotic effect resembles very closely natural sleep. It is applicable to acute hallucinatory conditions, insanities associated with rheumatism, and in the epileptic psychoses. In status epilepticus, per rectum it is one of the most valuable remedial agents. In some conditions, combinations of chloral with morphia are of much utility. Chloral is a heart poison, and its use is contraindicated in cardiac and vascular disease. Chloral-amid is of little value.

**Paraldehyde** is a simple hypnotic whose utility is not sufficiently appreciated. Naturally, its bad taste and the rather disagreeable odor left upon the breath have limited its sphere of usefulness; but it has no bad influence upon the heart or nutrition. It can be given in heart disease, and patients seem to thrive and grow fat upon it. The dose is from one-half to two drams, but increasing doses are necessary, and I have had patients who have taken four or more drams at a dose. It is especially useful in conditions of inanition and in insanities founded upon hysteria or neurasthenia. The taste and odor of the drug can be concealed in orange-water or weak brandy. Amylene hydrate is of less value; it stands between chloral and paraldehyde.

**Veronal, trional, and sulphonal**, as simple sleep-producing agents, are prominent where nothing but sleep is the object to be attained. Trional and veronal act quickly, sulphonal slowly; hence a combination of two of them in equal doses is particularly fortunate in its results, inducing, as it does, rapid and prolonged slumber. Five to ten grains each, or more, if indicated, may be given at bedtime with a glass of hot milk. The tastelessness of these drugs affords the possibility of administering them without the knowledge of the patient, mixed with salt or sugar, or spread with butter upon bread. Sulphonal used for a long period produces muscular weakness and incoordination. All of these agents may, after a time, give rise to some disorder of the alimentary canal. They are said to occasionally increase the intensity of auditory hallucinations.

**Moral Treatment.**—Psychotherapy is among the most important means of treatment of insanity. The general practitioner is especially concerned with it in the early stages of mental disorder; later, if the patient is turned over to the care of the asylum, it is still of the utmost importance, and the physicians in institutions know well the necessity and utility of moral agencies in effecting a cure or in at least ameliorating the condition of their charges. Physicians who have much to do with ordinary functional nervous disorders—hysteria, neurasthenia, mild depression, and hypochondriasis—are familiar with the wonderful influence they are able to exert over the mental attitude of patients thus afflicted, by kindness, patience, firmness, interest, and sympathy. Every-

thing they say or do, if rightly said or done, conveys a suggestion, inspires hopefulness, increases the efficacy of their prescriptions, points out the way to health and a new lease of life. The insane also are in the same way dominated by the personality of the physician and of those chosen to carry out his instructions. Some physicians are fortunate enough to possess peculiar gifts in this way, and their influence is potent for incalculable good. Aside from this personal influence, the physician is called upon to direct and regulate the entire disposition of the time of the patient and to make for him the environment suitable to his malady. He prescribes isolation from friends, the care of strangers, the rest-cure, the periods and kinds of exercise, the mental and manual occupations, the amusements, all of which go to make up psychotherapy. Some of the principles of this moral treatment we will now briefly touch upon.

The value of isolation in melancholia and of the rest-cure for both acute mania and melancholia has already been mentioned. There are cases of melancholia, however, in which a modified rest-cure is better than the complete rest-cure. In such cases, after recumbence in bed from six in the evening until noon the next day, much of the afternoon may be spent in simple exercise, such as walking slowly about out-of-doors. It is best not to seek, by amusements, visits of friends, and other cheerful devices, to rouse the melancholic from his depression, for usually these attempts rather add to his misery by force of contrast. A neutral atmosphere, so far as the emotions are concerned, is best, though an occasional word of confident reassurance is useful.

In acute stages of insanity it is best not to discuss the hallucinations and delusions of the patient, although neither physician nor nurse should ever fall in with or act upon his erroneous ideas. Whenever good judgment suggests, a brief but positive denial of the truth of the imaginings of the patient should be made. Later on such correction may with advantage be made more frequently and constantly.

When the patient is not taking a rest-cure, occupation of some kind is essential to his progress toward recovery. Most useful are all forms of muscular or manual employment, for labor of this kind keeps the attention more or less fixed upon what is being done, the flow of ideas is checked and limited to a considerable degree, and the mind is prevented from concentrating itself upon illusions, hallucinations, and delusions. Moreover, muscular exercise is an outlet for superfluous energy; motor excitement is reduced by it; tissue metabolism is accelerated; and when the work is over, the organism gains all the more readily a certain composure of mind and repose of body. Out-of-door occupation is best—garden and field work for men, garden work for women; walking, bicycling, etc., for either sex. Among indoor employments we have ordinary housework, drawing, knitting, sewing, embroidery, carpentry, wood-carving, etc., all of which employ the muscles methodically. In certain cases mental occupation is useful, though it should be of the simplest kind. For instance, during my practice at the Hudson River State Hospital for the Insane, we found much value in the establishment of a regular country school, attended



by patients of all ages. We had "spelling lessons," copying lessons, reading aloud, blackboard exercises, geography, simple arithmetic, singing, and so on.<sup>1</sup>

A very important point in the management of the insane is never to practise deception upon them in any way. Be absolutely truthful in every statement to them. Never remove a patient to an asylum under the impression that it is a hotel or sanatorium. It is better to state exactly what is going to be done, and then use force in the removal, if necessary.

Hypnotism has been frequently practised upon the insane, in the effort to modify hallucinations or delusions, rarely with any definite success, occasionally with ill results, and generally with no effect whatever.

There are a few conditions among the insane which require particular treatment or management. Among them are:

**Suicidal Tendencies.**—Suicidal patients are among those who require constant watching and the removal of every means of self-injury. This is often difficult in treating such patients in their own homes. How difficult, it may be conjectured from the fact that, even in asylums, with all their safeguards, suicide is by no means infrequent. Thus, 145 patients in the asylums of the State of New York committed suicide between October 1, 1888, and September 30, 1896.

Suicidal patients are to be watched night and day, and kept in bed, and even put in restraint, if desperate. I have known a patient to strangle herself with a cord while lying in bed under the eye of a nurse. Another, broke a small piece from a china plate and tried to cut her wrists under the bedclothes. While suicide is most common among melancholies, patients with general paresis, paranoia, epileptic psychoses, and toxic delirium sometimes attempt it. The physician attending such patients should see to the guarding of windows and the removal of keys, locks, scissors, weapons, drugs, strings, long pins, matches—in fact, of all instruments and means which he may suspect to be utilizable for a suicidal purpose.

**Refusal of Food.**—The acutely maniacal often can not be made to take sufficient nourishment, because they do not stop long enough in their ideomotor excitement to permit of eating. The watchful and persevering nurse can generally, by persistent effort, induce the patient to swallow a considerable quantity of liquid food (preferably in a metal or heavy china cup, because the patient frequently knocks the vessel from the hand of the nurse). Such patients can often be fed, as already stated, immediately after a hypodermatic injection of hyoscin or atropin before the supervention of sleep.

Other patients refuse to eat because of delusions of poverty or poisoning, suicidal proclivity, or simply from absolute distaste.

Where ordinary means fail, the nasal tube should be resorted to,—one of large caliber with rubber funnel attached,—and through this, once or twice daily, a mixture of a pint of milk, two or three raw eggs, a little meat-juice, and, if needed, brandy, may be introduced.

<sup>1</sup>See also *Psychotherapy*, page 771.

Before resort to this means nutritive enemata may be employed (three raw eggs, a half-pint of milk, a half-pint of water, and a little mint-juice).

I have been in the habit of delaying the use of the nasal or stomach-tube to the last moment of safety, even for several days, rather than subject the patient to the excitement of its employment. It is only in rare instances that feeding is not effected in some other way before the use of the tube becomes imperative.

**Violence and Destructiveness.**—Hypodermatic medication and hot wet-packs are indicated in periods of excitement with tendency to violence and destructiveness. It has already been intimated that active physical labor or exercise is a safety-valve for patients with proclivities of this kind. Isolation in an empty room with protected windows is sometimes resorted to in institutions, and abroad the padded room is a favorite place for patients whose violent propensities may lead to serious injuries to himself. The padded room consists simply of a room lined as to walls and floor with cushions. Mechanical restraint is used in the last extremity, when chemical restraint and other means have failed. The camisole and safety-sheet are employed only in cases with desperate suicidal tendencies, proclivity to excessive masturbation, great violence and destructiveness, and where needed to keep in place surgical dressings, splints, etc. In asylums mechanical restraint has been nowadays almost entirely abandoned.

**Masturbation.**—Masturbation is more often the consequence and concomitant of insanity than its cause. It may be ameliorated occasionally by drugs like bromide, camphor, and lupulin. Cold baths and hard physical labor are more successful in combating this habit. In excessive masturbation, constant watching day and night or the use of mechanical restraint is necessary. The use of blistering fluids on the genital organs is only of temporary service. There are instances in which the habit is so fixed and so uncontrollable—for example, among some imbeciles—that surgical interference would be quite justifiable (castration, clitoridectomy, ovariectomy, section of the pudic nerves, ligation of the vas-deferens).

## CHAPTER VI.

### MANIC-DEPRESSIVE INSANITY.

MANIC-DEPRESSIVE insanity is best exemplified by cases of circular insanity in which we have recurring cycles of maniac and melancholic outbreaks. But it is the merit of Kraepelin to have studied large numbers of patients suffering from mania or melancholia for periods of years, delving deeply into their previous histories, and following them up long after they had left his immediate professional care. This method of observation led to surprising results, not only in the matter of diagnosis, but also of prognosis. The profession had long fancied that little could be added to our clinical knowledge of the types of



insanity however much remains to be learned as to their pathology. Certainly, very little hard-work was needed to complete the maniacal picture, and melancholia has been a classical syndrome since the time of Hippocrates. That these two connected phases of mental disorder sometimes appeared successively in the same individual, as an alternating or circular insanity, was also well known, but such combined examples were esteemed exceptional and rare. It was here that the astute German clinician stepped in and, by taking the long view over periods of years in his cases, made the discoveries which have rather upset our former classifications, and overturned some of our criteria for prognosis.

He learned by his investigations that not only is alternating insanity much more common than psychiatrists deemed it to be, but that the majority of patients suffering from what we formerly looked upon as a pure mania or a plain melancholia, judged by their previous or subsequent histories to be subject to recurrences of attacks, which may be either maniacal or melancholic; and, furthermore, that there are many patients who present on careful study simultaneous manifestations of both these phases—i. e., a mixture of manic and melancholic symptoms at one and the same time.

For instance, the following table shows the contrast between the cardinal symptoms of mania and melancholia:

Mania.	Melancholia.
Excitation.	Depression.
Accelerated flow of ideas.	Retarded flow of ideas.
Motor excitement.	Motor inhibition.

Now we have occasionally cases of *manic stupor* (combination of exaltation and motor inhibition), or again *agitated depression* (combination of motor excitement, accelerated flow of ideas and depression), or still further the so-called *unproductive mania* (combination of exaltation, motor excitement, and retarded flow of thought).

The term manic-depressive insanity is thus made to cover almost all of the old types of insanity that we formerly looked upon as separate, sharply-defined clinical syndromes, viz.: mania, melancholia, and circular insanity. And Kraepelin naturally assumes for manic-depressive insanity an identical pathology for its contrasted or mixed manifestations, though what such pathology may be is wholly a matter of speculation. Kraepelin limits the term melancholia to the depressed psychoses of senility. There is no doubt that we owe Kraepelin much for the new views thus given us. It may be, however, that modifications will be made as time goes on in this conception of the manic-depressive syndrome. The brilliant exponent of manic-depressive insanity has the advantage of us at present in having already accumulated his material requiring decades of observation for verification, while we must wait years yet with our own cases before determining for ourselves the ultimate truth of his conclusions. Many of us doubtless have cases in mind which have passed through an attack of ordinary mania or melancholia with recovery, without history of previous attacks, and with no subsequent attacks for years to the present time. We must now leave the feel-

ing that recurrence in one or the other phase is a possibility, and yet the patient may go through life without such recurrence. Under the circumstances, is it worth while to change the name of the disorder from its well-known designation to the complex phrase manic-depressive insanity? And is it altogether profitable to exchange that classical immortal expression "melancholia" for the cumbersome phrase, "the depressed type of manic-depressive insanity," when we mean exactly what we did before, only assuming a new theory as to probable pathology? Why abandon the word "mania," when language needs condensation, for a phrase like "the manic type of manic-depressive insanity?" The clinical pictures remain the same; their interrelations are differently conceived. Some of the features of these psychoses which led Kraepelin to classify them under the new head of manic-depressive insanity, and to assume them to be equivalents of one and the same fundamental pathological process, are as follows:

The co-existence in the same subject of alternating mania and melancholia (circular insanity).

The presence in all forms, whether excited, depressed, or mixed, of certain fundamental symptoms, such as psychical inhibition (weakened attention, retarded flow of ideas, insufficient attention, indifference) and excited mental automatism (flight of ideas, irritability, impulses, delusions, hallucinations and illusions, fixed ideas, obsessions).

**Classification of Manic-depressive Insanity.**—Kraepelin divides the forms of psychoses under this heading as follows:

1. The manic type of manic-depressive insanity.
2. The depressed type of manic-depressive insanity.
3. The mixed type of manic-depressive insanity.

The manic type is separated into three forms: simple, delusional, and confusional.

The depressed type is divided into three forms: simple, delusional, and stuporous.

The mixed type is exemplified in cases of circular insanity, and also in very singular and varied mixtures of the two contrasting phases at the same time. Thus, some patients present a combination of psychomotor excitement with emotional depression, and others psychomotor inhibition with emotional exaltation. Still another group of cases is characterized by a combination of stupor with motor excitement.

A further classification of manic-depressive insanity, showing the various old forms included under this heading, is as follows:

- |   |  |
|---|--|
| 1. Periodic insanity.                       | Recurrent mania.<br>Recurrent melancholia. |
| 2. Alternating insanity. Circular insanity. |  |
| 3. Irregular forms.                         |  |

**Etiology of Manic-depressive Insanity.**—What has been said in the previous chapter as to etiology of insanity in general need not be repeated here. Kraepelin finds heredity a feature in about eighty per cent. of his cases of manic-depressive insanity. This psychosis forms about fifteen per cent. of all admissions to asylums. The disease gen-



usually begins before the twenty-fifth year, but a first attack may take place before the tenth year or not until after the fiftieth year of life.

**Prognosis.**—The prognosis of recovery from the existing attack of mania and melancholia is favorable, from circular insanity, unfavorable. But from the new standpoint, given us by Krapelin, we are not able to preclude a recurrence of attacks of mental disorder in a patient recovering from mania or melancholia. While the prognosis, therefore, is favorable as regards the single attack, the possibility of subsequent attacks must be carried in mind by the physician in discussing the prognosis with the family.

#### THE MANIC PHASE.

As already indicated, the cardinal symptoms of the manic phase are the elated mood, flight of ideas, and motor agitation. An outbreak of mania is often preceded by a period of depression lasting from a few days to a few weeks, sometimes as long as two months. This prodromal stage is characterized by a general feeling of malaise, vague uneasiness, and hypochondriacal complaints, accompanied often by headaches, cephalic paresthesias, constipation, loss of appetite, sleeplessness, and some loss of flesh.

When the true mental disorder begins to manifest itself, the sorrowful mood begins to give way to an exalted condition, which the patient looks upon as a state of renewed health and well-being. He takes a renewed interest in everything, and becomes unusually cheerful and talkative. The degree of increasing exaltation varies much in different cases. In mild cases the patient begins to surprise his intimates by his loquacity, frolicsome remarks, jocularity, and by his rather immoderate actions and undertakings. He enters upon many new schemes; makes innumerable calls upon friends and acquaintances; writes numberless letters; purchases unnecessary articles; and is inclined to excessive indulgence in tobacco, wine, and venery. There is considerable mobility or lability of the emotions, so that the elation may readily pass into conditions of anger or tears over trifles. In more severe types all of these symptoms are aggravated. A veritable chaos of ideas throngs through his mind, and the effects upon movement of this crowding series of ideas amount to a constant motor agitation. The patient laughs, declaims, sings, shouts, makes grimaces, dances, runs about, and becomes destructive and filthy, all inhibitory idea-associations ceasing to have any influence over the rising torrent of thought. In still severer grades we have the picture of an acute delirium, boisterous incoherence, a motor agitation attaining to violent jactitation, and an actual and considerable increase of temperature.

The patient with mania is fundamentally optimistic and egotistic. Everything about him is rose-colored. He feels rejuvenated; rejoices in his health, strength, and vitality; is delighted with the vivacity of his ideas and the untrammelled virility of his intellectual processes. His general and special sensibilities are ordinarily unaffected; in only about one-fifth of the cases are illusions and hallucinations present, and these are almost always limited to vision. Occasionally there are illusions

and hallucinations of taste and touch. Illusions of the special senses are more frequent than hallucinations. The inmates of extreme youth or age are especially prone to manifest hallucinations. Mania marked by the presence of numerous illusions and hallucinations is often designated as hallucinatory.

The flight of ideas in mania is naturally most conspicuous in the speech of the patient, which varies from garrulity to logorrhea. In the milder degrees of loquacity we are still able to follow the sequence of associations. The sentences are often bound together by the ordinary relationship and connections of ideas, but among which many latent ideas spring into consciousness and expression; and, again, the sound of words spoken suggest others of similar sound, giving rise to rhymes and assonances. Thus, the sight of the physician may suggest drugs, a certain apothecary, in a special street, in some familiar town; and the town may in turn give rise to another series. On the other hand, the physician's "How do you do?" may invoke a string of assonances (verbigoration) commingled with sentences expressing their associated ideas—*doe, two, new, grow, blue, crew, etc.* But in the more striking



Fig. 111.—Maria Pagan.

grades the logorrhea is so pronounced that it is impossible to find clues to any association, whether of sound or idea. It becomes a chaos of words, consequent upon an actual dissociation of the ideas in the rushing stream of thought—a secondary incoherence. The entire loss of inhibitory control of ideas is especially shown in the absolute lack of modesty, in the tendency to the employment of vulgar and obscene words and expressions. This profanity and obscenity become all the more astonishing by contrast, when it is observed, as it often is, even in the most refined and cultured of women.

The attention of the patient with mania is extraordinarily increased, so that the most insignificant trifle in his environment does not escape him. But this very increase of the power of attention, combined as it is with an unpausing stream of ideas, entails an absolute lack of concentration. His attention cannot be held a moment. It is constantly being drawn or distracted to every object or occurrence in his environment and we speak of this symptom as *distraetibility*. When the excitement is intense and the distraetibility high, there is often an appearance of clouding of consciousness and an apparent moderate disorientation, especially apt to be shown in the mistaking of persons. The rapid sequence of ideas tends to give them all an equal value (a leveling of ideas). The patient's memory, too, seems preternaturally intense, and it is remarkable how, after recovery, he may remember all the de-



tails of his delirious activity with great distinctness. Indeed, the patient, in the midst of the chaotic turmoil of his mind, often recognizes, as if he stood apart from and judged himself, the very silliness of his fancies and acts. The judgment-associations are, in fact, normal.

The elated mood and rapid flow of ideas give rise to delusions of expansive character, mostly in regard to strength, beauty, and intellectual powers, but often also in relation to wealth, social position, etc. In severe cases there are the most marked delusions of grandeur, the patient affirming himself or herself to be a prince, president, king, queen, Christ, the bride of Christ, the mother of God, etc. A peculiarity of these affirmations is their transitory character, their impermanence. A patient will, in the same breath, call himself a millionaire, broker, and king, and in the next a minister of the gospel and railroad magnate. If sharply told by the physician to stop such nonsense, he will often say it was only a joke, or he had said such things for fun. This shows very well the latest consciousness of the patient of the true state of affairs. Occasionally, but rarely, the delusions take on a paranoid character, and in a mild type of the disorder one might well confuse this phase with a genuine paranoid psychosis. When such a condition recurs from time to time, it has been called *paranoia periodica*.

The sexual instinct is morbidly exalted, giving rise in both sexes to immodesty and obscenity of speech and manner, and often to sexual excesses and masturbation.

The actions of patients with mania correspond in character to the degree of acceleration in the stream of ideas. When this is very great, turbulence, violence, and destructiveness are common, not with any homicidal or suicidal intent, because they are incapable of acts requiring any particular concentration of mind or reflection, but simply as the result of uncontrollable automatic impulses.

Sleeplessness is characteristic of this condition. General sensibility appears to be benumbed, probably because of the want of concentration of thought. Patients seem insensible to changes of temperature and to severe pain. Such a state often masks the most serious disorders, like pneumonia or the pains of labor. I once made an autopsy upon a woman suffering from acute mania who died suddenly. She had been for days in the wildest excess of mind and body. The cause of death was an acute peritonitis from rupture of a perforating duodenal ulcer. The peritonitis had evidently existed for several days, yet this painful affection had clearly had no effect upon the course of the mental and motor symptoms.

Some cases of mild maniacal character exhibit a peculiar tendency to logically explain and excuse their insane acts, and this type is often designated as *reasoning mania*.

As already stated, mania often begins with a prodromal stage of depression. After the exalted stage has subsided and at the beginning of convalescence, a reactive stage of depression is presented, characterized by irritability, sensitiveness, and lachrymity. This stage of depression may be so intense as to be an actual melancholic phase of simple nature or accompanied with stupor. In instances of this kind

the possibility of the patient's having the circular form, instead of a simple manic state, is to be considered.

Except in the severest type of mania (acute delirium) the bodily temperature runs a normal course, sometimes even showing a subnormal character. In acute delirium the temperature may reach  $104^{\circ}$  or  $105^{\circ}$  or more. The pulse is small and normal, or but slightly increased in frequency in mania. There are no paralyses, no true anesthesias. The absence of fatigue is often surprising. The deep reflexes are exaggerated, as a rule. The salivary secretion is frequently increased. Perspiration is diminished and sometimes transformed in character, so as to give a peculiar and often extremely disagreeable odor (akidrosis). Gastric disorders are nearly always manifested, and the tongue is heavily furred, frequently dry. In severe cases albuminuria, propeptemia, and hyaline cylinders are frequently found. The general bodily weight diminishes during the progress of the disease, but rapidly increases with convalescence. There is a corresponding condition of the appetite, an anorexia during the early stage and until the culmination; then an increase of appetite amounting often to bulimia. The face is sometimes slightly suffused, but, as a rule, marked by a yellowish pallor. As the patient exercises this becomes more noteworthy, and at the same time the features become pinched and sharp and the eyeballs sunken. This outline and color of the face, with a tendency to dryness of the lips and a heavily furred and dry tongue, are indications of the progress of exhaustion.

**Varieties.**—According to the intensity of the manifestations, upon the basis of the course of the disease, from the nature of certain concomitant symptoms, and, fourthly, in relation to some of the etiological factors, the manic phase is frequently divided into several varieties bearing some special qualification. *Mania mitis* is the mildest form of the psychosis. *Hypomania* designates also a mild type, and the so-called *remitting mania* is always of a hypomanic nature. *Acute delirious mania*, a very rare disorder, is possibly sometimes a most aggravated condition of the manic phase of manic-depressive insanity, but doubtless more often the result of some acute infection or intoxication due to actual unsurgical physical disease. The term *transitory mania* was formerly employed to describe a delirious condition of very brief duration, a few hours or a day or two, but these cases do not really present the characteristic symptoms of a true mania. *Periodic mania* is a form in which attacks of mania follow one another with perfectly normal but generally irregular intervals of days, months, weeks, or years. The attacks themselves last from a few days to a few months. Usually the prodromal depressive stage is absent, the culmination rapid, and convalescence seldom marked by the interesting depressive affects of ordinary mania. The periodic attacks are very apt to be distinguished by the presence of special symptoms, such as a reasoning tendency, tendencies to impulsive acts, arson, stealing, assaults, sexual and alcoholic excesses, and to severe headaches. The longer periodic mania endures, the less distinct become the normal features of the intervals. *Recurrent mania* and *intermittent mania* are only other names for periodic mania. Various etiological factors have in times past given rise to such designations as epileptic, alcoholic, morphia, puerperal, senile mania, etc., in some of



which the minimal excitement takes a special color from its cause. Thus, the toxic delirium are generally acute hallucinatory conditions.

**Course of the Disease.**—*Recovery* takes place in some seventy per cent. of cases. Sometimes it is exceedingly rapid, but usually the progress is gradual and rhythmical to the normal state. This rhythm is a sort of oscillation between good and bad days, but with constant improvement. Occasionally the patient improves steadily and uninterruptedly until recovered. Recovery is sometimes not perfect, so that we speak of it as recovery with defect.

*Death* takes place in but five per cent. of cases. The cause of death is sometimes exhaustion, as in acute delirium; more often an intercurrent affection, such as pneumonia, nephritis, and the like. Heart disease and alcoholism add greatly to the danger of lethal termination.

**Diagnosis.**—One must be careful not to confound delirium from fever with an attack of acute mania. Except in acute delirious mania, the absence of fever in the mental disorder should be distinctive. The three cardinal symptoms of mania should be kept constantly in mind—*viz.*, the exalted mood, the accelerated flow of ideas, and the motor excitement. We must determine whether the syndrome is complicated by other conditions, such as general paralysis and alcoholism. A very mild degree of mania may pass unrecognized, unless it is possible to diagnose it from a pronounced change in the character of the individual and from the accompanying insomnia. The physical symptoms and the defect of intellect should suffice to distinguish the exalted stage of general paresis from an acute mania. The delusions, too, of paresis have a peculiar monotony of character that differentiates them from the exalted ideas of the maniac. Sometimes, however, there will be difficulty in making a speedy diagnosis between these two analogous exalted conditions.

The manic phase must be differentiated from conditions of excitement often found in dementia praecox. If the characteristic deterioration of the latter disorder is not observed, long observation only can insure the correct diagnosis.

**Treatment.**—What has already been said in the chapter on General Treatment is applicable here. The requisite isolation and supervision of a patient with acute mania can seldom be satisfactorily accomplished outside of an asylum, unless his means are sufficient to secure the needed nurses and suitable surroundings.



Fig. 334.—Manic phase of long duration.

Rest in bed aids in the prevention of exhaustion, and renders supervision, care, and feeding more easy. To induce sleep and allay motor excitement, hydrotherapy and the hypodermatic use of hyoscin, hyoscyamine, or dibosin are extremely valuable. In mild cases equal parts of trional and sulphonal are preferable to the drugs just mentioned. Paraldehyd is also an excellent hypnotic for mild cases.

Overfeeding is also an extremely important indication. Liquid and easily digested foods are to be recommended. The bowels should always be regulated. Brandy is added to the liquid food when exhaustion is imminent, but otherwise stimulants are contraindicated. Should there be danger of collapse, the repeated hypodermatic injections of ordinary salt and water (ten to fourteen ounces) over the abdomen or in the thigh are valuable. In the depressed period of convalescence small doses of opium are often useful.

#### THE DEPRESSIVE PHASE.

The cardinal symptoms of the depressive phase of manic-depressive insanity are, as stated above, a depressed mood, retarded flow of thought, and motor inhibition.

The affective state in this psychosis varies from simple dejection, in which every thought and everything in the environment of the patient has a sorrowful color, to a state of profound depression, in which the patient is either paralyzed by the dreadful nature of his concepts or thrown into a state of agitated suffering associated with marked preconscious distress. There are many degrees lying between these extremes. This morbid depression is in many ways paralleled by and analogous to the conditions of normal grief in which we observe a varied behavior of different individuals under the influence of distressing emotions; some become strangely quiet and still; others, again, make noisy and agitated demonstrations of their grief. Normal grief, too, is often accompanied by sensations of choking and of sinking at the heart, which are similar but comparatively mild manifestations of the preconscious anxiety and dread of the psychosis. We observe often in melancholia a rhythmic oscillation of the state of depression during the day, and frequently from one day to another. Thus, the depression is at its height in the morning (when suicidal tendencies not infrequently present themselves), being followed by a recession with another exacerbation toward night. Very often patients sleep better on alternate nights, and manifest intense emotional depression on alternate days. In some cases, presenting what is



Fig. 20.—Depressive phase.

known as the apathetic form of melancholia, the patients complain that they have no feeling at all; that they are affected neither by things cheerful nor grievous, pleasant nor painful; that they have no love for family or home, or interest in anything; that they can



never be sad or glad again. Sensory disturbances are often absent. In the apathetic variety there may be analgesia. Marked illusions and hallucinations are observed in only about a tenth of all cases of melancholia. The paresthesias in the region of distribution of the vagus are neither illusions nor hallucinations, but they may give rise to delusions; they depend probably upon vasomotor disturbances. The melancholic perceives and identifies ordinary and special sensations slowly and with difficulty. The peripheral stimuli of his environment go unnoticed. When hallucinations are present, they usually affect most of the senses, and are terrifying and dreadful in character. The patient sees the flames of hell, phantoms and ghosts of dead persons; hears voices which reproach and threaten him, or the sounds of machinery and other tortures which are being prepared to cut him up or mutilate him; smells and tastes horrible things, and so on.

Next to the effect of depression, the most noteworthy symptom of melancholia is the slowing of the thought processes. Sometimes the retardation is altogether out of proportion to the depression, the retardation being great and the depression very mild.

The processes of memory are retarded, and the attention of the patient difficult to gain. A minute or several minutes are required for the answer to the simplest question. Sometimes no answer is given at all, or at most the lips stir inaudibly.

The contents of the concepts may, in milder degrees, show no delusions. More often the patient attempts to explain his feeling of abject misery and distress either by the presence of some fancied physical ailment (hypocondriacal melancholia, with delusions of having syphilis, consumption, cancer, impotence, incurable disorders of the stomach, bowels, etc.), or as the result of some sin of his past life. To the delusion of having sinned an especial color is given by the character of the patient's early education. Thus, a strong religious bias gives rise to delusions of having committed the unpardonable sin, of being doomed to hell, to everlasting punishment, to be buried alive, etc. Often such delusions are connected with some trivial error of his past life. For instance, a patient of mine recently told me, "I once chloroformed a dog to death and buried him. I think now I made a mistake in not making positively sure that the dog was dead, and as a result I am doomed to be buried alive also, and to be tortured with dreadful thoughts through eternity, each day the torture growing more dreadful, up to the decilicath power of intensity."

Patients often say they are not sick, they are only wicked. They have committed sins not only against God, but against society. Not only must they undergo the punishment ordained by Heaven, but they must answer to man for infringements of human law. They are to be put in prison, to be killed, to be hung. Thus they come to delusions which are somewhat similar to persecutory ideas in that they believe the officers of the law are after them, etc. These differ, however, from the true persecutory delusions in which patients have no self-depreciatory ideas, but believe themselves to be the innocent victims of diabolical conspiracies. Delusions of poverty are very common, but more especially in involution melancholia, to be described later on.

The conduct of the melancholic depends upon the contents of his consciousness. In his expression are note the lines of extreme depression, or of fear and terror. The patient with the delusion of sin or poverty, for example, presents motor inhibition. He sits in one place with head bowed down, unmindful of what goes on about him, indifferent or apathetic to all questions put to him, resisting every attempt to give him food or medicine, or to dress and undress him, or to give him exercise. He is lost in the contemplation of his misery. Another patient, with these or similar depressed ideas more accentuated, or with marked hallucinations, will wring his hands, tear his hair, walk or run up and down, bewailing his misfortunes, or seeking to escape the dreadful life in store for him. In the first case the motor inhibition may be so complete as to make the patient perfectly immobile, so that not a single voluntary movement is made; even micturition and defecation are involuntary. Suicidal tendencies are observed in every type of melancholia, but especially in those with precordial distress and agitation. In the milder degrees, an attempt at suicide is often the first intimation to friends of the actual existence of insanity, since in these cases, outside of the sorrowful mood of the patient, the intellectual processes may go on as before. Cases of melancholia attonita (with marked motor inhibition) also often make attempts at suicide, unexpected explosive attempts, the result of the sudden letting up of mental and bodily tension. This has been called the *explosus melancholicus*. Homicidal attempts and violent assaults are occasional in melancholia. A melancholy mother kills her children to put them out of an unhappy world; or a sudden dangerous assault is made as an explosion of motor tension. Hypochondriacal melancholics may mutilate themselves. Patients with melancholia have also been known to enter upon alcoholic excesses to drown their misery. The refusal of food is almost the rule of conduct in all forms of melancholia. Sometimes this refusal rests upon a deli-



FIG. 106.—Depressive phase.

delusional foundation; the patient thinks he cannot digest his food, that it never passes through him, that he is too poor to pay for it, that he is too wicked to eat, that he must do penance, and so on. Or he refuses food with deliberate suicidal intent. Generally, personal anorexia, constipation, and gastro-intestinal disorders are at the basis of this refusal to eat.

The pulse is usually subnormal in frequency, though sometimes, especially in agitated forms, accelerated. The peripheral arteries are contracted and the extremities cold. The respiration is retarded and superficial, as a rule, though it may be increased in the agitated types. Sleep is much disordered, and even altogether absent, in severe

cases. The patient emaciates both through refusal of food and because of disordered digestion. The gastric juice and saliva are often diminished in quantity. The tongue is foal and furred, and obstinate consti-



pation is present. As a result of constipation, elevations of temperature may be observed, but otherwise the temperature is undisturbed. The surface temperature in the extremities is often much reduced. Amenorrhoea is frequently induced by the depressive as well as the manic phase.

**Varieties.**—The depressive phase may show itself as follows:

1. Mild depression, with very light retardation.
2. Strong depression, with very light retardation.
3. Depression with the development of delusions of a depressed nature, and various grades of retardation. While the delusions are usually melancholic, they may also be paranoid or hypochondriacal in character.

4. Complete retardation, with the facial expression of depression (depressive stupor).

5. Periodic, recurrent, or intermittent types. These designations have the same significance as in mania, attacks of the depressive phase occurring at intervals throughout the life of the patient.

**Course of the Disease.**—There is no such distinct prodromal stage in this phase as in the mania. The period of invasion is deliberate, and the symptoms chiefly manifested at first are gastro-intestinal disorders, dyspepsia, loss of appetite, constipation, accompanied by sensations of pressure in the head or headache, insomnia, and general malaise. The depression itself is the cardinal early psychic symptom. This phase, like all psychic disorders, is slow in its progress, and runs a course of from three to six months in the most favorable cases, but sometimes a year or two or three elapse before recovery takes place. Ordinarily, recovery is gradual, and is frequently accompanied by a species of reactive exaltation. Occasionally recovery is quite rapid. In women the approach of convalescence is indicated by a return of the menstrual function. In all cases improvement in physical health accompanies convalescence.

Recovery from the attack takes place, according to various authorities, in between 70 and 80 per cent. of the cases, but of course the tendency to recurrence in the same or opposite form in all these cases must always be kept in mind.

Death in cases of melancholia is due to suicide, marasmus, visceral disorders, diarrhoea, pneumonia, etc. A very large number of long-standing cases die of tuberculosis.

**Diagnosis.**—One of the most common conditions with which melancholia may be confounded is a depressed stage of general paresis. The chief points of distinction are the actual intellectual defect nearly always demonstrable in paralytic dementia, and especially the physical symptoms of paresis, pupillary changes, facidlingual tremor, characteristic speech, greatly exaggerated or lost deep reflexes, and one-sided facial weakness. The depression of the paralytic dement is superficial. His melancholy delusions are ordinarily distinguished by their incoherent and preposterous character, by the absurdity of their contents. In addition to these points, the signs of previous syphilis and the age from thirty-five to fifty years would have some corroborative value in the diagnosis of general paresis.

The *stupor* of one form of dementia præcox must be differentiated from that of the melancholic phase. In the former we have, with the stupor, often tension and negativism, and the face is either expressionless or grinning. In melancholic stupor the facial expression tends to show depression or suffering.

There are instances of such a disorder as typhoid fever being temporarily mistaken for melancholia, but naturally the course of the temperature and the character of the stupor or delirium would soon correct such an error.

**Treatment.**—The first consideration in the treatment of acute melancholia is isolation. Separation from the friends and relatives and removal from the environment in which the psychosis has developed are of the greatest importance. With familiar faces and objects about him, and with his kin offering their help and sympathies, the keenest realization of his condition is brought home to the melancholic. He feels among them all the more deeply a sense of his incapacity, of his inability to fulfil the ordinary duties and demands of his usual daily life. Whether the patient is to be isolated by commitment to an asylum depends upon several circumstances: his means; the intensity of his malady; the presence of suicidal tendencies. There are very mild cases in which moderate travel, a sojourn in the country with a nurse, a few months at the house of some country physician or in a small private asylum, will result in recovery. But the responsibility for such a course must rest with the physician who advises it, and he must keep in mind the danger of suicide in even the mildest type of melancholia. Not a few lives have been needlessly sacrificed by the inexperience of the consulting physician. Besides extreme watchfulness on the part of the caretaker, who is not to leave the patient alone either night or day, a modified or a complete rest-cure is to be undertaken. For mild degrees of melancholia rest in bed from 6 P. M. until noon of the next day, with plenty of out-of-door exercise during the remainder of the afternoon, is most commendable. For the more severe types, continual rest in bed is requisite. The food should naturally be easily digestible and assimilable, and the patient should be made to take considerable quantities of milk and milk products (koumiss, matzoon, *smal*, etc.), raw eggs, meat-juices, and stimulants, when these are indicated. Massage and general faradization (sufficiently strong to contract the muscles) are useful to take the place of exercise in cases taking the complete rest-cure. Constipation should be regularly counteracted by abdominal massage, frequent purgation, glycerin injections, enemata, etc. This is particularly necessary in cases suspected of suffering from auto-intoxication. In these cases, too, gastro-intestinal antiseptics—such as salol, gr.  $\gamma$ , or beta-naphthol, gr.  $v$ —should be administered three daily two hours after eating. Twenty grains of glycerophosphate of soda in a large glass of hot water a half hour before eating is also a useful remedial agent in melancholia. For sleeplessness the prolonged warm bath or the hot wet-pack is to be recommended; in the event of their failure to induce a few hours' sleep in each twenty-four hours, sleep-producing drugs are necessary. Sulphonal and trional, of each



five to seven grains, given together at bedtime with a glass of hot milk or a cup of hot soup, are efficient in mild cases.

The opium treatment is a sort of specific for melancholia, especially when there are agitation and precardial anxiety and distress. Beginning with a medium dose three or four times a day, we gradually increase it as required. Laudanum—the solid extract—or codein may be administered by mouth. When employed hypodermically, which is usually best, the watery extract of opium is used. It is preferable to administer morphin only in the most aggravated cases, and in these it may often be advantageously combined with hyoscin, hyoscyamin, or duboisin. It is needless to say that the opium treatment should not be made known to the patient, and it is carried out with more safety, as regards the formation of a habit, when the patient is in an institution. As the patient improves, the opium is gradually reduced until it can be finally cut off altogether. Opium does not increase constipation, except possibly for a few days when first employed; it seems actually in many cases to diminish it. Sometimes, indeed, we need to treat diarrhoea that arise as a result of the opium treatment.

As soon as it becomes possible to do so, physical occupation should be begun and encouraged. A life out-of-doors, made interesting by different kinds of amusement or labor; walks, field studies in natural history (botany, ornithology, geology, physical geography, etc.), golf, bicycling, agriculture, and gardening—all of these have their place among the remedial agents at the disposition of the discerning and judicious physician.

#### THE CIRCULAR OR ALTERNATING TYPE.

This form, now included generally under the designation manic-depressive insanity, has for decades been recognized as a special type of disorder characterized by the periodic alternation of states of melancholic depression and maniacal excitement. Among its well-known names are alternating insanity, insanity of double form, insanity of double phase, circular insanity, and cyclic psychosis. It is the perfect type to which the term manic-depressive insanity can be given without question or argument. There has been criticism of the inclusion of the old syndromes of simple and recurrent forms of melancholia and mania, just described under so comprehensive and general a head. The criticisms have been based upon the following:

Kraepelin and his followers are chiefly familiar only with the severe types of psychoses met with in asylums. They seldom come in contact with the numerous mild cases of mania and melancholia observed by the specialists outside of institutions, where single attacks of one or the other disorder may occur but once in a lifetime.

Even his adherents see many patients with recurrent melancholic attacks only, and without manic phases or symptoms, and vice versa.

The symptoms of mania and melancholia, being so directly opposite in character, would rather argue a possibly different seat of the disorder in the brain, and a possibly different pathological cause. Since we know nothing whatever either of seat or cause, it is rather wild speculation to assume the same pathology.

If Kraepelin had been content with enlarging the domain of circular insanity by the inclusion in it of his mixed cases and all of the recurrent types of mania and melancholia that show a tendency to alternation of phase, criticism would have been less.

It at least has simplified diagnosis to include every possible maniacal or depressed psychosis under the one term, and it was a rather singular hesitancy to exclude involution melancholia (all the symptoms of which are often met with in younger individuals without involution) for so long a time; but the need of consistency seems to require including that under the general head at last.

Probably there will be no more classification upheavals until we are able to classify *de senibus et senium iaculorum*.

**Etiology of the Circular Type.**—Heredit plays an especially significant part in the causation of circular insanity (60 per cent.). Not



FIGS. 35, 36.—A case of circular insanity, photographed first in maniacal or a calm phase, and some months later in the melancholic phase (Dr. Arzoo).

only do we find in the family history of the majority of these cases hereditary equivalents of different kinds, but direct inheritance of this particular variety of mental disorder is strikingly frequent.

Many degenerates exhibit a tendency to an alternating variation of mood. Sometimes they are depressed and sometimes cheerful. It is probable that this oscillation of moods in an individual with strong hereditary taint may be the rudimentary foundation upon which the superstructure of a circular insanity is subsequently laid.

**Symptomatology.**—The symptoms will vary at any given time according to the phase which the disorder has reached at the time of examination—the phase of depression or the phase of exaltation. The *melancholic period* may present any one of the forms just described, from



a simple depressed condition, scarcely distinguishable from the normal state of the patient, to the most pronounced melancholic syndrome. In some cases we have melancholia simplex, in some agitated, in others stupor. When, in any given case, the melancholic phase recurs again, it is prone to wear the same features as in the first attack. Thus, mild depression or simple melancholia, melancholia agitata, or stupor may reappear again and again as the cycle returns, with the same phase and character over and over again. While this is true in the majority of cases of circular insanity, it is not always so, for occasionally the recurring depression changes its type in the various sequences. As intimated in the pages on the depressive phase, there is often a species of reactive exaltation in the convalescent stage of the disease, and occasionally this reaction becomes so accentuated as to develop a maniacal condition, so that we have presented to us a picture very like that of an alternating insanity.



Fig. 100.—Scheme of course of disease in periodical circular insanity.

Like the melancholic phase, the *maniacal period* of circular insanity may vary in character from a condition of mild exaltation and exultation to the severest types of maniacal excitement and incoherence. As in the depressed period, there is the same tendency of the maniacal phase in its recurrences to present regularly the identical features of former attacks, though there are also exceptional instances here where subsequent outbreaks wear a different maniacal aspect.

In the article on the mania phase mention is made of the fact that the convalescence from that psychosis is not infrequently characterized by a reactive depression, a laryngeal irritability. In some instances this may attain to the degree of a true melancholia, and thus place before us a cycle similar to that of an alternating insanity.

Ordinarily we recognize two degrees of intensity in circular insanity—one in which both the mania and melancholia are mild, and one in which both phases are severe. But there are mixed types, in which the mania may be mild and the melancholia severe, or vice versa.

Mild types of circular insanity—instances in which both the depressed and exalted phases are so moderate in degree as not to permit of commitment to an asylum—are not infrequently met with by the practitioner, and they are often difficult cases to handle properly. Thus, I have in mind two brothers, not over fifty years of age, who are both afflicted with circular insanity, manifested in a form very distressing to the relatives. A description of one will describe the other, and not only him, but many other similar cases:

E., male, aged fifty-four, single, with hereditary taint, has for many years been subject to alternating attacks of depression and exaltation. I have seen and examined him in both phases. There is little, if any, discernible interval, but a gradual merging of one phase into the other. The depressed period lasts for from three to six months. In this, his expression is dejected; he feels that life is a failure, that he can not live long. He consults various physicians for different maladies which he thinks may account for his general malaise. He can not concentrate his mind on anything, can not read or write letters; refuses to transact the most necessary business in connection with his estate. He talks little, and broods over the mistakes and follies committed in the exalted phase of his disorder. He is rather suspicious and distrustful of his family. Sometimes he is inclined to put an end to his misery by suicide. Little by little this weight of depression begins to lighten, and he passes insensibly into a condition in which he begins to feel himself rejuvenating. Life takes on a little rosier color; his malaise vanishes, and a sense of well-being begins to infuse itself through his body. His expression changes from the fixed look of deep dejection to one of cheerful variability. In the place of quiet brooding we note an awakening interest in things about him. He begins to talk vivaciously, to be facetious and jolly, to write letters to his friends, to make frequent social calls, to take up the threads of affairs. He discards the doctors, for his health and strength were never better. He takes up some of his old hobbies, one of which is the collection of antiques, arms, plate, furniture, pictures, and specimens of ceramic art. He spends money freely, rather too lavishly. His collections are gathered together in storage warehouses, clubs, his own home, and the houses of his friends. He becomes extravagant and wasteful; enters on great schemes of money-making, in which he becomes interminably entangled and meets with financial losses. His friends expostulate, and he becomes irritable and angry. He leaves them, to live in hotels. He buys a pair of fast horses and takes a drive of several weeks all over the country for hundreds of miles around. He grows boisterous in his conversation, neglectful of the ordinary courtesies and civilities of social life, is lavish in his invitations, becomes a little excessive in drinking, is restless both night and day, travels from one city to another on the most trivial and eccentric errands. He sleeps little. Endeavors on the part of relatives to check the anomaly of his conduct bring from him threats of suits and of personal violence, and letters which are quarrelsome, offensive, even profane. With all this, there is no intellectual defect. He never has actually attempted any overt act which



would put him under the control of the law, or aid in his commitment to an asylum to save the dissipation of his energies and the waste of his property. Any jury would discharge him, for his conversation would show good memory, active intelligence, keen-witted replies to all questions. Step by step this stage of exaltation begins to pass away. He sinks nearer to his normal level, resumes a more natural conduct toward his family and friends, until again the depressive elements reappear in his mental condition. Each stadium lasts for from three to six months, so that the cycle fills about one year.

**Varieties.**—There are two main varieties of circular insanity. One is a true circular insanity in which the phases follow each other in a perfect cycle thus: mania, melancholia, mania, melancholia, mania, melancholia, and so on. The other type is one in which there is a certain periodicity of the maniomelancholic attacks as follows: mania, melancholia, interval, mania, melancholia, interval, mania, melancholia, interval, etc. Most cases can be catalogued under one of these two headings, but there are deviations which do not exactly conform to these well-defined types, and some authors have attempted to make further, but it seems to me unnecessary, subdivisions, upon the basis of variations in the length of interval and irregularities in the sequence of the phases. Thus, some authors divide the varieties as follows:

1. Circular insanity, with the following sequence: Mania, melancholia, mania, melancholia, etc.
2. Alternating insanity, with this sequence: Mania, interval, melancholia, interval, mania, interval, etc.
3. Insanity of double form, with either of these two sequences: Mania, melancholia, interval, etc.; melancholia, mania, interval, etc.
4. Alternating insanity of double phase, with the following sequence: Mania, interval, mania, interval, melancholia, interval, melancholia, etc.

**Course of the Disease.**—In some patients circular insanity has its inception in the melancholic period, and in others it begins with the maniacal phase. Usually the initial stadium is melancholia. The transition from the depressed to the excited phase and vice versa is sometimes astonishingly sudden. The period of transformation may occupy but an hour or even less. In most cases the merging of one period into the other is very gradual. Another and extremely rare mode of transition is by successive alternations of depression and exaltation, an oscillating or rhythmic transformation. Still another method of change is by means of a lucid interval, brief or long, between the alternating phases, thus: mania, interval, melancholia, interval, mania, interval, melancholia, interval, etc.

There is extreme variability in the duration of the maniomelancholic cycles. Sometimes they exhibit great irregularity of interval, from a few days to a year or more. Sometimes the maniacal phase lasts one day and the melancholic one day, so that the cycle is completed in two days. In other cases, again, the cycle is completed in two weeks, or a month, or a year. Where alternation is completed in short

periods, there is a tendency to great regularity. Usually the melancholy phase lasts longer than the maniacal.

**Treatment.**—All cases of circular insanity are best treated in an asylum in order to prevent suicide in the melancholy phase, and violence, excesses, and riotous extravagance in the maniacal period. Unfortunately, it is not always possible to protect the patient by this means, since juries are prone to allow every man his freedom, no matter how dangerous to himself or others, so long as he does not believe as a raving maniac before them. Even in the intervals of lucidity it is better for the patient to be under medical supervision in some institution, with the hope that the disorder may be arrested and future cycles prevented or postponed by the treatment. This treatment is based upon the principles described in the chapter on Treatment and in the pages on the manic and depressed phases in this chapter.



Fig. 112.—Scheme of course of disease in continuous circular insanity.

The rest-cure and hydrotherapy are recommended for both phases of the cycle. Hyoscin, hyoscyamin, and atropin (gr.  $\frac{1}{16}$  to gr.  $\frac{1}{4}$ ) are useful in the excited stage, and the opium treatment in the depressed stage.

### INVOLUTION MELANCHOLIA.

The term involution melancholia has been employed for the depressed psychosis that usually begins in women at the climacteric, between the fortieth and fifty-fifth years, and in men after the fiftieth year. The Manich school until recently made this form a clinical entity, distinct from manic-depressive insanity, and excluded it, together with certain other rather ill-defined depressed states called *symplocotic depressions*, from the larger category. But in 1907 Dreyfus published his investigation of the histories of eighty-one cases of involution melancholia from the material of the Heidelberg asylum, and concluded that nearly all were actually cases of manic-depressive insanity, since the func-



mental symptoms of this latter disorder were present in the great majority. The conclusions he drew have been accepted by Kniepelin and many of his followers. For the sake of greater simplicity, and not to complicate for the student and general practitioner elements that are still debatable and full of confusion for psychiatrists, involution melancholia is here placed under the caption of manic-depressive insanity. Indeed, if the term manic-depressive insanity is to cover all that its originator claimed for it, it might well cover a disorder such as this, in which we have no symptoms that are not to be found in the melancholias of other periods of life. While anxiety, for instance, is common in involution depression, it occurs at all ages, and, on the other hand, retardation, one of the cardinal symptoms of the depressed phase of manic-depressive insanity, is also observed in the involution psychosis. The three general and usual characteristics of this type of melancholia may be set down as depressed mood, fear, and anxiety. Doubtless, in so far as the physical changes incident to the retrogressive period of life are present in the patient, they modify or color the symptoms manifested.

**Symptomatology.**—The prodromal symptoms are similar to those that precede the development of the depressed phase of manic-depressive insanity, as already described under that heading, possibly somewhat more deliberate in their evolution, owing to the age of onset. When the disorder is at its height, which is reached by gradual stages, we have, first, a profound depression, more or less stationary, shown in a sad or suffering expression, and in gentle weeping or loud wails of despair, with wringing of the hands, etc. Secondly, this general depressed state may be interrupted by anxious states, gradually increasing in intensity, or the change to the agitated condition may take place so suddenly as to be a *typus melancholicus*. Ordinarily, the agitation shows itself in an unrest, varying in degree from restless walking to and fro to incoherent running up and down, tearing the hair, and beating the breast. The anxiousness is almost always of the persecutory variety. Thirdly, we have the development of delusions usually of sin or of unpardonable sin, sometimes of poverty, more rarely of reference. In addition to this ordinary delusional content, we may have added thereto delusions of a hypochondriacal or markedly paranoid nature, though these last are never systematized. Fourthly, in a goodly number of cases we note the presence of psychomotor inhibition, as described in the pages on manic-depressive insanity. Fifthly, illusions and hallucinations of sight and hearing, sometimes of taste and smell, are frequently manifested, and are especially important in the anxiety states, because of the dangerous and violent outbreaks they may induce.

In all these cases there is a strong tendency to suicidal attempts, and the refusal of food is practically a constant symptom.

Orientation, intellect, and memory are undisturbed, except in cases that go on through exhaustion into conditions of confusion or where they merge into stupor.

These cases are usually divided into *melancholia simplex*, where there is neither delusion nor agitation, only a profound state of depression, *melancholia anxiosa* or *agitata*, in which the anxiety is predominant, and

*melancholia stuporosa*, in which the psychomotor inhibition is the striking feature. To these qualifications are added hypochondriacal, religious or persecutory, according to the delusional content presented.

**Course and Prognosis.**—The involutional period of life influences the recovery-rate in this type of melancholia, so that fewer of these get well. Some forty per cent. recover fully. A certain number improve, some become chronic, and a majority of the rest die of some intercurrent disease, especially tuberculosis. Suicide terminates the existence of a considerable number.

**Treatment.**—Nothing need be added here to the general indications for the treatment of such cases given in the chapter on Treatment, and in the part devoted to the depressive phase of manic-depressive insanity. To protect from suicide by anxious supervision, to feed despite the refusal of food, to overcome insomnia by hydrotherapy, and, if need be, drugs, and to prevent exhaustion in the anxious cases, these are the main points that require consideration.



## CHAPTER VII. DEMENTIA PRAECOX.

**DEMENTIA praecox** is a disease beginning usually in early life, and characterized chiefly by a more or less marked and peculiar enfeeblement of the mind, but manifesting upon this basis a considerable variety of symptoms, such as emotional indifference, weakness of judgment, flightiness, verbiage, automatic obedience, cataplexy, echopraxis, stereotypy, negativism, mutism, impulsive actions, affectations, grimaces, and unemotional laughter, delusions of a depressed or grandiose nature, and hallucinations.

It is not easy to offer a brief and clear definition of dementia praecox and I have made the above from an analysis of Kraepelin's descriptions of the multifarious phases of this psychosis. He has brought together under this name a group of mental disorders, the distinguishing feature in all of which is a special type of dementia most clearly outlined in terminal conditions.

There are cases in which all of the psychical functions are equally enfeebled, as in the disorder which we have been accustomed to call primary dementia, and which type is now included in the new category. These are exceptional. The characteristic of the enfeeblement of mind in dementia praecox is the inequality of weakening of the several faculties, a sort of selective deterioration.

Kraepelin includes in this large group of cases our old class of cases known as primary dementia; the catatonias of Kahlbaum, the hebephrenias of Kahlbaum and Hecker, and the large group of cases that we in this country have been in the habit of calling chronic mania and chronic melancholia according to the nature of the delusions presented (grandiose or persecutory) and which are held by Kraepelin to be paranoïd types of dementia praecox. True paranoia, or chronic delusional insanity, is not included.

**Classification.**—This author, therefore, divides his group dementia praecox into three types of the disease, viz.:

1. The hebephrenic type.
2. The catatonic type.
3. The paranoïd type.

Other authorities have made a still further division into five classes instead of three, as follows:

1. Hebephrenia.
2. Hebephrenia.
3. Catatonia.
4. Paranoïd.
5. Mixed forms.

It must be borne in mind that these types are more or less interrelated and often run into each other.

**Etiology.**—Dementia praecox, according to Kraepelin, represents some fourteen to fifteen per cent. of all admissions to asylums, of which

five to six per cent. would represent the hebephrenic and catatonic types and nine to ten per cent. the paranoid group. In the statistics of the New York State hospitals for the year ending September 30, 1909, the total number of admissions of insane persons was 5142, and the diagnosis of dementia præcox, and states allied to dementia præcox was made in 1281 of these cases, nearly twenty-five per cent. In the statistics of Welfsohn, of the Zürich asylum, dementia præcox constituted nearly thirty per cent. of the admissions. This disorder is essentially a disease of early life. In a study of 296 cases Krapelin found that sixty per cent. began before the twenty-fifth year, though typical examples may be noted at any age. In the earlier years the disease is more apt to appear as a simple, gradually progressive dementia (hebephrenic type), a little later in adolescence as an acute or subacute form with catatonic symptoms, and still later as a paranoid condition. The two sexes are equally liable to the disorder, though there are more males affected by the hebephrenic form and more females by the catatonic and paranoid forms. Heredity was noted in seventy per cent. of the cases, and physical stigmata of degeneration are frequently to be observed. In ten to eleven per cent. of Krapelin's cases the development of the disease was preceded by severe acute diseases, such as typhoid fever and scarlet fever, but, as usually, years had elapsed between the two episodes little value is to be attached to such relations. Alcoholism seems to have no place as a cause. It is noteworthy that more than six per cent. of Krapelin's cases developed in prison.

As to the pathological basis for dementia præcox, Krapelin is of the opinion that we have to deal with an actual chemical injury to the cortical cells causing their deterioration or destruction, and that the origin and development of the psychosis are best explained by the theory of an auto-intoxication arising possibly in connection with processes going on in the sexual organs. He is led to the last idea by the unusually close association of the disease with the age of development, with menstrual disorders, and with pregnancy and childbirth.

A theory evolved by Jung on the basis of the Freud psychology is that many cases are of psychogenic origin—that is, that they owe their origin to repressed emotional complexes, and, to account for the actual deterioration, he conceives that some toxin is ultimately created by the emotional condition.

Another view which has perhaps wide credence is that the original impetus to psychical development implanted in an individual becomes prematurely exhausted, and the intellectual powers give out, dwindle, and disappear.

**Symptomatology.**—Let us first take a view of the general symptoms of dementia præcox and later seek a mental grasp of the syndromes presented by the three types mentioned above. The underlying characteristic of *psychical infatuation* common to all has already been dwelt upon.

The simple perception of external impressions is little, if at all, impaired. The patient apprehends all that goes on about him far more than one would suppose from his behavior. One is surprised to learn



that patients apparently wholly stupid and dull will take note of innumerable remarks and events in their neighborhood, suddenly revealing their lucidity by some apt observation.

In consequence of this, orientation is seldom disturbed in dementia praecox. As a rule, the patient knows where he is, recognizes those about him, and clearly appreciates time.

*Hallucinations and illusions* are common, especially in acute or sub-acute conditions. The auditory are most frequent, after which come hallucinations and illusions of sight and common sensation (feelings of currents passing through the body, of being touched, or of influences). These sensory symptoms tend at first to be of unpleasant nature and to distress and depress the patient, but are later received with unconcern, or at times with amusement as if they were a species of theatrical performance.

*Consciousness* is in many cases fairly clear throughout, though generally somewhat clouded in excited and stuporous states. On the other hand, the faculty of attention is regularly subject to serious disturbance.

One of the most prominent symptoms is the *failure of interest*. Though the patient perceives what takes place in his environment, he contemplates all with indifference.

*Memory* is relatively little impaired in dementia praecox, except after the lapse of years, when some reduction in the faculty may be noted.

There is a *refractoriness to the flow of thought*, so that ideas are associated with difficulty even to the extent of an incoherence in which no possible connecting link can be perceived. But it must be conceded that this incoherence is often more apparent than real. It is very common to observe stereotypy of language, the senseless repetition of phrases for days, weeks, months, playing upon words, rhyming, and other forms of reiteration.

The faculty of *judgment* is affected in all cases. Transitory or lasting *delusions* develop in a very large number of cases. In the early stage of the malady the delusions are of depressed character, hypochondriacal, melancholy, persecutory, but later on, grandiose ideas appear, either in association with the depressed ideas or displacing them. Such delusions, as a rule, are transitory, changeable, silly, and senseless, apparently because of the rapid advance of intellectual weakness. In the paranoid type they are less transitory and variable, but even here grow more and more disconnected and less prominent as the disorder progresses.

There are, as a rule, profound and striking disorders in the sphere of the *emotions*. An anxious or sad state marks the beginning of the disease in an extraordinarily large number of cases. Rarely there is mirthfulness with constant laughing at this early stage. But more important than these transitory conditions of depression or exaltation is the invariable development in this disorder of *emotional deterioration*. This, indeed, is one of the fundamental characteristics of dementia praecox. The *failure of interest* already alluded to depends in a measure upon the general emotional decadence, since the faculty of attention has an emotional foundation. The patient feels neither sadness nor joy. He is

indifferent to his relatives and friends, to his old occupations and pleasures, to pain, to fears, to desires. He is insensitive to injuries, to bodily discomfort, to uncomfortable physical positions, to pricking with a needle, but not always to hunger. The presence of food will often arouse an interest that is otherwise wholly lacking.

This emotional indifference, together with deterioration of will-power, gives rise to numerous morbid manifestations in the domain of conduct. The patient is often practically will-less and has lost every ordinary incentive to normal behavior. He sits stupidly about, negligent of person and dress and of the demands of nature, or at times develops some sudden impulsive motor activity which may reach the degree of a stormy manual excitement. Impulsive actions, silly and senseless, often dangerous and destructive, are manifested, but wholly without will or purpose. They are the planless expression of an inner tension. Very often the automatic impulse will be reiterated over and over again in either language or conduct, constituting *stereology*. The patient will repeat the same words or phrases over and over again, or turn backward and forward or round and round, or move the hands to and fro, or take attitudes for hours. A very singular symptom in this connection is *negativism*. Every incentive to some sort of motor expression is met and overcome by a counter-incentive, by an inhibition. The will is blocked, as Kneplin describes it (*Willensperren*). To the category of negativism belong mutism; resistance to feeding, dressing, undressing; obstinate resistance to all that physician or nurse tries to do for the patient; the hobbling in of the saliva and excretions; the creeping under the bedclothes; the turning away when spoken to; the inaccessibility to all requests and influences. Negativism varies in extent and degree in various patients and often in the same patient. It is not uncommon for a condition of negativism, quite unresponsive to outward influences, to be broken through by some sudden senseless motor impulse arising in the patient himself. After the impulse has been executed the patient relapses as suddenly into the former resisting state.

With such reduction and perversion of the will in dementia precox it is not surprising to observe at times in every case some degree of *negative automatisms*, such as echolalia, echopraxia, and catalepsy.

Dementia precox is a psychosis that surprises the student and general practitioner by the singularly capricious and bizarre symptoms usually presented. They are altogether outside of his experience. In melancholia, for instance, we ordinarily observe only great exaggerations of psychological functions that are normal—a depression that goes beyond normal bounds. In mania, especially in the mild forms, the normal flow of thought, normal motor activities, and normal feeling of well-being are simply exaggerated, but to a degree that we recognize as pathological. In certain paranoic conditions and in paranoia the speech and conduct may be most of the time normal. It is in dementia precox that the extraordinary happens—the sudden motor explosions, impulsive actions, irrelevant laughter, stereotypy of speech and movement, negativism, catalepsy, echolalia, echopraxia, neologisms, strange imperative ideas, the symptom of thought deprivation, and the salutatory fancies, affecti-



tions, mannerisms, and so on. Now, doubtless, all of these curious phenomena have their psychological explanation if we could only get at them. But much of the time such patients are practically inaccessible to us, and it is only at intervals and by the most careful study and research that we are occasionally enabled to obtain a glimpse of what goes on in their minds. There has been a great deal of investigation of the psychology of dementia præcox of late, and much that is interesting has been discovered, and many psychological theories have been propounded. The Krapelin psychology is chiefly concerned with the idea of emotional deterioration, a selective dementia. Strinsky, studying the problem on this basis, and finding constantly, especially in early stages, an incongruity between the emotions expressed and the ideas apparently behind them, formulated his theory of "intrapyschic staxis," meaning simply thereby an inco-ordination between ideation and emotional tone, which leads to all the peculiarities of emotional expression and to peculiarities in the stream of thought, as evinced in incoherent speech and writing, with their stereotypes, assonances, etc. This inco-ordination may, however, be more apparent than real, for so difficult is it to gain access to the mind of the patient that we are not absolutely sure yet that the emotions are actually deteriorated, nor can we say with any certainty what ideas rising from the sub-conscious is connected with the emotional expression we have just witnessed. It may be a normal co-ordination of thought and emotion, for aught we know. Weygandt, looking upon diminished power of attention as the chief symptom of the disorder, prefers the designation "apperceptive dementia," meaning thereby a dementia in which the power to take in, digest, and add to the intellectual store is lost. This would amount practically to a reduction in consciousness, almost a species of dream-state, and thus account for many symptoms like automatisms, stereotypes, etc. Gross has proposed the name "dementia sejunctiva" for the disease, based on his theory that we have in the disorder a disintegration or *sejunctio* of consciousness. He explains that such disintegration of consciousness would mean the simultaneous flow of functionally separated series of associations. This would naturally lead to broken and disjointed associations, unrelated to each other, reaching consciousness in a bizarre manner, and thus give rise to such symptoms as autochthonous ideas, sudden impulses, hallucinations, ideas of thought-domination, imperative ideas, thought deprivation, salutatory fancies, etc. Pelletier finds an analogy between recovery and dreams and dementia præcox, in all of which conditions we have lowered attention and a shallow and sluggish flow of associations. Jung<sup>1</sup> has written an excellent monograph on the psychology of dementia præcox, in which he discusses critically the work of psychologists in this field to date, and then builds up a parallel between this disorder and hysteria, and applies the principles of the Freudian psychology to dementia præcox. He assumes many cases of this disease to be of psychogenic origin, to depend upon emotional complexes, and he makes a brilliant analysis of a case of the paranoid type as a paradigm.

The physical disorders of dementia præcox have no pathognomonic

<sup>1</sup> Jung, C. G., "The Psychology of Dementia Præcox," New York, 1909.

value, though many have been studied and described. Among them are attacks of syncope; epileptiform, apoplectic, and hysterical seizures; localized spasms; tetany; transitory paralyses; aphonia; singultus; choreiform movements; "athetoid staxia"; apnea; exaggerated reflexes and increased mechanical irritability of the muscles; dilated pupils usually; vasomotor disorders (cyanosis, edema, dermatographia, hyperidrosis); increased salivary secretion; pulse slow or rapid, often weak and irregular; subnormal temperature; anorexia; diffuse enlargement of the thyroid gland; menia; emaciation in acute and subacute conditions, but rapid increase in weight in later stages.

Having thus passed over cursorily the general symptomatology of dementia praecox, it remains to place before the student rather briefly the main features of the three marked types described by Kraepelin. The term *hebephrenia* has been applied to the mildest degree and moderate affective forms of dementia praecox, but the difference is so slight, being one merely of degree, between it and hebephrenia, that it hardly merits any fuller description here.

#### THE HEBEPHRENIC FORM.

This type is also called *dementia praecox simplex*. It is a simple, progressive enfeeblement of the mind, varying in degree, and manifesting itself as a subacute, sometimes as an acute, psychosis. The earliest symptoms are almost neurasthenic in character, such as insomnia, headache, inability to work, and general feelings of fatigue and malaise. Gradually a change in character takes place. The patient becomes solitary, brooding, quiet, and indifferent to family, friends, recreations, and studies. Occasionally suggestions of delusional ideas are observed, depressive or hypochondriacal, ideas of being worthless, lost, going to pieces; and at this stage the disorder is often looked upon by relatives as perversity of disposition rather than as disease. Paranoid ideas may be exhibited, suspicious, ideas of reference, of being watched, of being drugged, but always in a more or less rudimentary way. Gruesome ideas may come to the surface at times. There are often occasional single or solitary hallucinations. Sometimes the beginning is very similar to hysteria, so that the differential diagnosis is difficult. Again, the onset may either take on a strong depressive color or, on the other hand, a mildly hypomanic character. Single catatonic symptoms are sometimes noted, and moderate negativism, minor mannerisms, and affectations. Very characteristic and frequent is laughter without adequate cause. The further progress after the initial stage is manifested by growing apathy, emotional indifference, increasing loss of will-power, and defective judgment, but without any striking loss of orientation or clouding of consciousness. Secure insight is often shown in the early period, the patient feeling that he is ill and changed from what he was before, and then expresses hypochondriacal ideas. The old memory-store is unaffected, but the power of adding new memories to it gradually grows more and more defective. The emotional indifference grows apace, and is most noteworthy in relation to friends and relatives.



There is often an increased appetite and the patient is prone to grow stouter with the progress of the disease. The course of the disease is one of years, though at times the dementing process proceeds very quickly. Partial recoveries and remissions take place in a small percentage of cases. The simulation by this form in the early stages of neurasthenia, hysteria, and the two phases of manic-depressive insanity must always be borne in mind in establishing the diagnosis.

#### THE CATATONIC FORM.

This type is distinguished as a progressive dementia, with the addition of peculiar conditions of stupor or excitement, and with symptoms of negativism, stereotypy, and suggestibility (echolalia, echopraxis, waxy flexibility). When Kahlbaum first described his syndrome of catatonia, he included under that caption cases of melancholia with stupor, stuporous conditions, and acute dementia.

The catatonic form is more common in women than in men, and is especially apt to set in after child-bearing. The prodromal symptoms and onset do not differ materially from those of the hebephrenic type. At first there are only now and then real catatonic manifestations, but as the disease progresses these become more and more manifest, with alternations of catatonic excitement and catatonic stupor.

Catatonic excitement may develop suddenly, even sometimes out of the stuporous phase, and is marked by stereotypes, impulsive actions, mannerisms, negativism, great motor excitement of a bizarre or rhythmic nature, and verbiage. Such patients often declaim theatrically, are destructive of clothing and objects in the room, resist violently any effort at control, and are filthy to a degree. Often there are marked sexual excitement, open masturbation, and obscene speech (coprolalia). Sometimes the excitement takes the form of an ecstasy. Occasionally it has a strong superficial resemblance to the psychomotor exaltation of a manic attack.

Catatonic stupor may also develop suddenly in the midst of an excitement just described, or gradually in the course of the psychosis. Fully developed, it presents mutism, negativism, and tonic or negativistic muscular tension. The muscles of the mouth are often contracted and protruded forward in a peculiar way, so that the name *Schisslmausspiz* has been given to this symptom. The face is mask-like, but may be played over by stereotyped grimaces. The fingers and thumbs are strongly contracted into the palms. Patients in such condition have absolutely no reaction to stimuli, even painful pricks with a needle, and require to be fed forcibly and cared for in every way.

The stupor may be suddenly transformed into excitement, or there may at any moment be a sudden explosion of extraordinary conduct or violence. There is a tendency to an irregular alternation of these phases in the course of the disorder. In less extreme cases we have moderate excitement or moderate stupor, varying with intervals when the chief symptoms are mannerisms, affectations, simple and quiet stereotypes, echolalia, echopraxis, etc. These conditions often last for long periods

of years. Hallucinations and transitory delusions of almost any variety are at times manifested. Very characteristic in this form are the remissions which may take place and last from a few hours to several weeks.

In diagnosis it is necessary to differentiate the occasional manifestations of catatonic symptoms in paresis, in epileptic states, in hysteria, in infection and imitation psychoses, in involution melancholia, and in senile dementia, and to distinguish the excited period from a manic phase. The mental enfeeblement is our strongest aid.

#### THE PARANOID FORM.

In hebephrenia and catatonia hallucinations and delusions are episodic and transitory, subordinate to the simple enfeeblement in the one type and to the catatonic syndrome in the other. The paranoid type is characterized by a rapidly developing mental weakness with preservation of lucidity, and by a chronic delusional and hallucinatory state.

Prodromal symptoms and onset are analogous to those in the other forms, but paranoid ideas are prone to develop quickly. They are never so elaborately systematized as in paranoia proper, though they may become more or less fixed and unchangeable in some cases, while in others they are often changeable, and of senseless and fantastic character. In many cases we observe symptoms reminiscent of the catatonic syndrome, and it is only the predominance of the delusional and hallucinatory symptoms that establishes the particular type. Besides paranoid ideas, we meet with grandiose delusions of an impermanent nature, and sometimes with hypochondriacal concepts. Neologisms and confabulation are often observed in this group.

Krapelin separates paranoid dementia into two main types:

1. Dementia paranoides, with permanence of countless, disconnected, constantly changing persecutory and grandiose ideas, associated with moderate excitement.
2. A type characterized by singularly fantastic delusions, innumerable hallucinations and illusions, a more connected development, lasting a few years, and then terminating in confusion.

**Diagnosis as to All Forms.**—Since dementia precox is essentially a disease of youth and adolescence, the majority of cases beginning between the eighteenth and twenty-eighth year of life, this is the first thought to come to the mind in the presence of a psychosis at this epoch of life. This age practically excludes forms of insanity like paresis, involution melancholia, and true paranoia (not usually fully developed till after thirty years of age). Other conditions that might come in question rarely would be hysterical and epileptic states and manic-depressive insanity. These can be excluded by a careful study of the symptoms and by the history of the patient.

**Prognosis in All Forms.**—Since this is a dementing psychosis, the prognosis must be considered unfavorable from the first. At the same time a certain number do recover, and many improve, and a certain number have remissions, as in general paresis. Remissions seldom last long, the disease recurring in a short time, to remain permanently.



**Treatment of All Forms.**—It is possible that in time we shall become able to recognize precocious tendencies in childhood, and learn methods of physical and mental training and psychotherapy that will help us to ward off in many cases the impending disaster. Certainly prophylaxis will have a great application here some time that it has not now. This would be of special value in the psychogenic cases. As to medicinal and other treatment, according to the symptoms presented, the reader is referred to the special chapter on Treatment. The fact that many of these cases are of perhaps psychogenic nature should lead us to be very careful in the early stages not to submit the patient, if it can be avoided, to the mental ordeal of asylum life, with its constant and suggestive contact with other patients, often worse off than himself. Isolation from the family will doubtless be advisable, but, if possible, among sane people, and where he can be given an occupation and exercise cure, which I have seen do more good in these cases than anything else. Such a system of cure should be in the hands of some intelligent and enthusiastic physician in the country. It is to be hoped that the urgent demand for home and family life with a physician in the country, who will take one or not more than two patients needing such care under his supervision, will be met ere long by an adequate supply. This system is well developed in England, but with us is practically non-existent.

## CHAPTER VIII.

### SENILE DEMENTIA AND OTHER SENILE PSYCHOSES.

"**DEMENTIA**" is a term employed to designate simply a general enfeeblement of all the mental faculties. It is often used improperly by the laity as synonymous with insanity. But in medicine it signifies only a general weakening of a mind once normal. Hence it is not applied to congenital mental weakness. The term "idiotcy," with its various degrees, includes all of those congenital psychic defects. There are innumerable gradations comprised in dementia, from the merest dulness to profound deficiency or complete loss of all the intellectual faculties. Such enfeeblement of the mind may be the result of serious cerebral diseases or disorders, such as epilepsy, alcoholism, syphilis, etc., when the dementia is qualified as epileptic, alcoholic, syphilitic dementia, etc. It is sometimes, though rarely, a sequel to acute insanities, like mania and melancholia, and to chronic psychoses, like circumscribed insanity and paranoia, and hence the distinctive term *secondary dementia* applied to such examples. It takes the chief part in the syndrome of paresis, so that that disorder is often entitled "paralytic dementia." Progressive mental enfeeblement not infrequently accompanies senile involution and organic changes in the brain incident to that epoch of life; hence the well-known disorder called *senile dementia*. Finally, there is a form of mental disease characterized in the main from the very beginning by selective psychic enfeeblement, and this malady is classified as dementia precox.

## SENILE DEMENTIA.

This is a progressive mental enfeeblement at the period of senile involution, dependent upon organic changes in the brain; therefore, a chronic organic psychosis. When the disorder begins before the sixtieth year and depends upon premature senility, it is sometimes called *dementia senilis parva*.

**Etiology.**—Hereditv has been noted in some fifty per cent. of the cases. Males and females suffer about equally. The disorder rarely appears before the sixtieth year. Mental stress and physical illness, together with the senile involution, are the chief etiological factors. In most of the cases arteriosclerosis takes part in the causation of the disease, inducing, as it does, general malnutrition of the brain, as well as frequent local degenerations of small or large extent.

**Symptomatology.**—Like general paresis, senile dementia may manifest itself in various ways, under the semblance of maniacal, melancholic, hypochondriacal, or paranoid types of psychoses, or by a simple progressive weakening of the mind, with only episodic appearance of delusions and hallucinations. The earliest symptom is failure of memory. The most recent memories disappear first in a sort of chronological order. After a time the patient fails to recognize any of his surroundings or any of the people about him. He converses with those near him, and miscalls them, as if they were old friends of long years ago. He lives over old events as if they were now enacted. Later on even these old memories vanish also. With failing memory, the judgment-associations perish. The patient commits many breaches of decorum, and later, with the degeneration of ethical feelings and the ascendancy of coarser instincts, may become very negligent, indecent, and unclean in habits; may pilfer and destroy things; may expose his person, masturbate, or attempt liberties with little girls, etc. His loss of judgment may induce him to foolishly squander his money and properties.

Illusions and hallucinations begin to manifest themselves. They are usually of terrifying character.

Delusions make their appearance. These are usually always persecutory in nature, and arise either as primary ideas or as the result of depression or on the basis of hallucinations. Next to delusions of persecution in frequency, we observe hypochondriacal delusions, with contents modified by the weak-mindedness present. Delusions of approaching poverty are quite common.

The underlying mood is often melancholic; an excited mood is extremely rare. Changeability with irritability is perhaps the most usual affective condition.

The behavior of these patients in relation to night is noteworthy. Illusions, hallucinations, delusions, and emotional states all become more pronounced at night. A striking feature, too, is extreme motor restlessness, especially at night. These patients try to get up from bed, to wander about the house, to get away from something or somebody. Sometimes true melancholic anxious states come on and lead to attempts at suicide.

So far as bodily symptoms are concerned, we note foremost among them a general senile decrepitude, to which are added senile tremor of



the hands, and often various stigmata of focal lesions in the brain (aphasic and jampaphasic attacks); sometimes hemiparesis, monoplegia, hemiplegia, apoplecticiform and epilepticiform attacks complicate the picture. The patients often complain of severe pains all over the body, of vertigo, ringing in the ears, sparks before the eyes, etc. Often, too, there is noticeable diminution of sensibility to touch and pain in various areas, or over the whole body.

Intolerance to alcohol and a tendency to drunkenness are often early symptoms in a beginning senile dementia. The course of the disease may be divided empirically into three stages—an initial stage, a stage of well-marked general dementia, and the end stage.

In the initial stage, which begins insidiously with a gradual decline of psychic functions, we note first the weakening of memory and the inaccessibility to new ideas and impressions, together with a slow change in character, manifested by irritability, egotism, excitement over trifles, easy weeping, childishness, and perhaps indecent and lascivious conduct (offenses against morals, exhibition, assaults on children). Often there is a condition of marked depression, showing itself in the form of a *senile melancholia* or a *senile hypochondria*. Sometimes the disorder begins with depression and stupor, on which basis grows gradually a *senile paranoid condition*. More rarely there is a hypomanic state (*senile mania*). Whatever the character of onset, or psychotic coloring, in all these conditions the chief feature is the falling power of retention and adding to the memory-store.

In the second stage the power of retention is wholly gone, and the patient lives only in years long past. Vocabulary and store of ideas gradually fail more and more, and disorientation as to time and place is complete. Still, during the daytime, despite these changes, the patient may automatically carry on his old life, read his papers, converse with family and friends, carry himself well, go to his meals regularly, play cards, etc., but often at night he is restless, sleepless, completely disoriented, sometimes delirious and hallucinated, with amnesia for these experiences when the day comes again. At times we meet with confabulation, and a condition reminding one of the Korsakoff psychosis. This state is sometimes designated as *senile Korsakoff's psychosis*.

The third stage is that of profound dementia, terminated by general physical disintegration and death from disorders incident to old age.

**Course and Prognosis.**—Senile dementia develops gradually upon the basis of senile psychic degeneration, and lasts, ordinarily, from three to ten years, sometimes with remissions which are never so noteworthy as the remissions of paralytic dementia. In rare instances an acute course is taken, the disease terminating by death in a few months. Paralytic attacks are not infrequently observed in the course of the malady, giving it a certain analogy to paresis. The prognosis is unfavorable, as the disorder is incurable and progressive to a fatal end.

**Diagnosis.**—The most important indications for diagnosis are defects of memory and judgment and acts dependent upon loss of ethical feeling.

**Pathological Anatomy.**—We observe at autopsy chiefly the following conditions:

1. Osteophytic deposits on the inner surface of the skull.

2. Pachymeningitis hæmorrhagica interna (more frequently even than in paralytic dementia).
3. Opaque and thickened leptomeninges.
4. Increased fluid, subdural, and in the meshes of the pia-arachnoid.
5. Distention of the ventricles with serum, and granular ependyma.
6. Extreme narrowing of the cortex, with gaping sulci.
7. General endarteritis deformans (often with foci of softening and hæmorrhage).
8. Wide-spread degeneration of ganglion-cells and association fibers.

**Treatment.**—Many cases of senile dementia can be treated at home. It is only when tendencies to suicide, sexual immoderacies, waste of property, and great ideomotor excitement are exhibited that commitment is necessary. The bromids are the best hypnotic for these cases. Paraldehyd is extremely useful, too, since it is efficient as a hypnotic and does not injure the circulation or affect the digestive apparatus. In melancholic phases opium acts well. Hyoscin and its congeners are not to be recommended because of their depressing action on the heart.

#### OTHER SENILE PSYCHOSES.

Some of these have already been referred to above. Besides the dementia which may be an evidence of premature senility, we sometimes have a *pseudo-paranoid condition* developing before the sixtieth year, especially in women. It begins like other paranoid conditions, finally developing into a psychosis with delusions often of a peculiar and extraordinary nature (colored thus by weakness), and in content persecutory, though sometimes delusions of infidelity are present. The persecutory ideas never become fixed or systematized, but are remarkable for their changeability and evanescence. They do not affect the conduct of the patient, and are often overrighable by argument, but only to return in some other form. The condition tends to become chronic and without dementia. Recovery almost never takes place.

We may meet with various alcoholic psychoses after the age of sixty. I have seen a considerable number of cases of *epilepsia tarda* after that age, sometimes with mental symptoms. It is not infrequent to meet with psychoses that are the result of organic diseases of the brain in old age, associated, for instance, with hæmorrhage and thrombus. It must be remembered, too, that real dementia præcox, manic-depressive insanity, and even paresis may at times develop in the period of senility. It is not uncommon to meet with infection, exhaustion, and toxic deliria in old age.

### CHAPTER IX.

#### PARALYTIC DEMENTIA.

**Synonyms.**—Dementia paralytica; Progressive general paralysis; General paresis; General paralysis of the insane.

**Definition.**—Paralytic dementia, as its name implies, is a disorder characterized chiefly by progressive enfeeblement of the mind, together with a progressive general paralysis of the whole body. It is essen-



tially a cortical disease, but its symptomatology is frequently modified by spinal complications. The psychic symptoms, in addition to the characteristic progressive dementia, present multifarious phases, neurotic, hysterical, hypochondriacal, melancholic, manic, circular, paranoid, etc. An expansive phase with delusions of grandeur is very common at one period or another in the course of the malady.

**Etiology.**—Intellectual overwork or strain, working on a foundation of syphilis, may be said to be the chief cause of general paresis. Heredity is not so important a factor in this disorder as in other types of mental disease. The rôle of heredity has been computed at from 10 to 40 per cent, and organic cerebral disorders in antecedents are more frequent than ordinary psychoses. No doubt the part played by heredity is larger in juvenile paresis. As regards sex, it may be stated that on an average males are affected two to five times as frequently as females. The disproportion seems to be less in the lower classes. As to age, the great majority of cases fall between thirty-five and forty-five years, and more between forty and forty-five than in any other period of five years. But general paresis may be encountered at any age, in the first decade of life or even occasionally in the seventh, though uncommon in both these extremes. It is a common disease in the great centers of civilization, where the intellectual stresses are most severe, and is comparatively infrequent among lower races, such, for instance, as the natives of Egypt and other parts of Africa and of many parts of Asia and the islands of the South Seas. There are even wide differences in the percentages of prevalence in various European states, the ratio being about the same in France, England, Italy, Austria, Switzerland, Holland, and the United States, but extremely low in some countries, such as Norway, Spain, and Turkey, as though for some unknown reason there were a racial difference in vulnerability. The disease appears to be proportionately more frequent among men of ability in business or professional life than among the ignorant and uncultured, which gave rise to the famous dictum of Krafft-Ebing that "civilization and syphilization" are the chief causes of paresis. The part played by alcohol is somewhat uncertain. There is often a definite alcoholic history antedating the development of paresis, and quite frequently it is a result or symptom of the disease. Very likely the rôle of alcohol is that of reducing vitality and general resistance, thus establishing a better soil for the action of the toxic germs. Intellectual overwork and strain perhaps act in like manner in impairing the vitality of the higher nervous structures. Trauma of the head, sometimes alleged as a cause, doubtless acts in a similar way.

Syphilis has now been demonstrated to be the essential factor in the causation of paresis, owing to more vigorous investigation of histories and clinical symptoms and the introduction of valuable laboratory aids to diagnosis. The spirochetes have been found in the brain and spinal cord. Paresis is a syphilitic disease closely related to tabes, which is also parasyphilis. While this is now accepted by all the leading authorities there are many features in the production of paresis by the specific germ that are still obscure—the wide divergence in susceptibility among various races, the selective determination of late nerve syphilis in some cases lightly affected with syphilis and the escape of so many de-

quite severe tertiary symptoms, the special virulence of some infections as if there were different strains of luetic germs. For instance, paresis in both husband and wife is not infrequent, and there have been cases published of five men infected by the same woman all developing paresis, and of three infected the same night by another prostitute, two later developing paresis and one tabes.

**Symptomatology.**—The disease is best studied in its three stages—the prodromal period, the established disorder (which may be exalted, depressed, or hallucinatory), and the terminal period of dementia.

**Prodromal Period.**—General paresis is one of the most insidious forms of insanity as regards its gradual, almost unnoticeable onset. Very often this early stage presents symptoms which lead to its being mistaken for neurasthenia. Indeed, the earliest symptoms may be neurasthenic in character, or even a combination of hysteria with neurasthenia. Sleeplessness, tremor, irritability of mood, hypochondriacal depression, dull headache, ophthalmic migrain, pains in various parts of the body, general malaise, loss of appetite, and digestive disorders—these are the manifestations which may be readily misinterpreted as purely of functional nature. It is only when other symptoms in addition to these are presented that a suspicion of a more serious malady may be entertained or the diagnosis actually established. These symptoms are, on the mental side: little faults of memory; errors in speech or writing; the misuse of words; the leaving out of letters, syllables, or words, or their reduplication in writing; growing indifference to the higher sentiments; loss of the critical faculty; small lapses in the proprieties, and failure of interest in the more important affairs of life. As these mental features become more and more pronounced, the patient loses and mislays things, makes mistakes in money matters, errs in appointments, confuses persons and objects, forgets his way, becomes easily angered, markedly offends the proprieties, shows extravagance in the use of money, evinces distinct loss of the ethical feelings, exhibits peculiarities to sexual and alcoholic excess, and becomes negligent of his dress.

In the earlier period the patient, like any neurasthenic, has a distinct consciousness of his own illness and observes his symptoms. But with the progress of the malady—and it is in this that we find an important contrast to the course of neurasthenia—he loses that sense of being ill, takes no further notice of his own symptoms. On the physical side there are a number of significant marks which are helpful in making an early diagnosis: defective innervation of one side of the face, causing a slight paralysis; transitory ocular palsies, diminished sensibility to pain, Argyll-Robertson pupils; diminished, lost, or exaggerated tendon-reflexes; a dark, pale, greasy complexion; lack of facial expression; jerky tremor of the faciolingual muscles at the beginning of voluntary movement; slight difficulties of articulation; rushings of blood to the head, and attacks of syncope or of mild or severe epileptiform convulsions. A number of other early symptoms have been described by various authors to which some value attaches: loss of tendency of localization of tactile sensations (*Zichen*); loss of the cremasteric reflex; testicular insensibility; peculiar respiration, with



short inspirations, followed from time to time by prolonged sighing expirations (Régis); gastric and vesical crises (Hurd); calcification of the sternum, with incurvation of the xiphoid appendix and consequent interference with thoracic breathing (Régis).

**Period of Establishment of the Disease.**—When the disorder is fully established after a prodromal period which may range over months or years, it is marked by both physical and mental symptoms which are usually characteristic:

**Chief Physical Symptoms.**—(1) Peculiar articulation and writing—the “paretic speech” and “paretic writing”; (2) tremor; (3) papillary disorders; (4) lost or exaggerated tendon-reflexes; (5) muscular weakness; (6) apoplectic and epileptiform crises; (7) emaciation; (8) trophic disorders.

**Mental Symptoms.**—(1) Failure of memory for both recent and old events; (2) diminishing number of concrete, abstract, special and general ideas; (3) weakening of judgment; (4) loss of sense of time and place (lack of orientation); (5) delusions (marked by enormous exaggeration, whether exalted or depressed); (6) hallucinations and illusions; (7) emotional irritability; (8) exalted, sometimes depressed, mood; (9) loss of ethical and esthetic feeling.

We will now examine these symptoms somewhat in detail.

The paretic speech is so characteristic that, heard a few times, it is always remembered; yet it is difficult to describe. There are shades of difference in various individuals, so that authors qualify the disorder of speech as drawing, stammering, hesitating, scanning, spasmodic, ataxic, and so on. It has some resemblance to the speech of a drunken man. Doubtless the main seat of the lesion affecting the speech of the paretic is in the cortical motor speech-center, but sometimes the lesion is probably in the bulbar centers connected with the elaboration of the motor impulses requisite to articulation. The jerky tremor or ataxia of the speech-muscles, together with incoordinated impulses from the cortical motor speech-center, is responsible for the peculiarities in speech. Labials and certain consonants are the most difficult for the paretic to enunciate, and the typical speech is shown in the attempt to pronounce such words or phrases as “electricity,” “artillery and cavalry brigade,” “immovability,” etc., in which the consonants may be left out, drawn out, mis-placed, or even reduplicated thus: “electricity,” “artillibillery,” “bigrade,” “immobility.” As the disease advances, the words are run more and more together, until finally the speech is utterly incomprehensible.

The handwriting of the patient is of equal, and in the earliest stages even of greater, importance. Lapses of words, repetitions of words or even sentences, and especially elisions and reduplications of letters or syllables are extremely significant.

The handwriting, as time goes on, becomes more and more tremulous, finally deteriorating into an illegible scrawl, but the chief points in paretic writing, often at a very early stage, when it is one of the most important diagnostic symptoms, are the elisions, reduplications, and sometimes substitutions in the spelling. For instance, one of my patients, a well-educated government official, who showed no neurological or mental symp-

toons, presented the following peculiarities in spelling in his correspondence, although his handwriting itself was still excellent:

"At the theatres the actors and actresses and other sorts of other amusements."

"Could play his eighteen holes like a man of twenty years of age."

"You remember that trip we made from Manila to Hong Hong."

The diagnosis of paresis was made on this characteristic writing alone. There was no history of syphilis, but the serology was corroborative of the diagnosis.

The tremor in parietic affects all parts of the body, but is especially noteworthy in the face and tongue. In the tongue it often takes on a fine, fibrillary character. It is very rare in even pronounced neurasthenic conditions to observe tremor of the facial muscles. Still we do meet with it at times, and the distinction that I would draw between the facial tremor of profound neurasthenia and that of paresis is that in the latter disorder there is a peculiar jerkiness and anxia in the tremor, especially at the beginning of a voluntary movement. Thus, in asking the parietic to wrinkle his forehead, an ataxic tremor will be set up in the occipitofrontalis. In snarling up the nose, it is observed in the small muscles about the cheek and nose. In showing the teeth, the ataxic tremor becomes marked in the levators of the lip. In protruding the tongue, there is a rapid, jerky tremor at the beginning of the movement.

As regards the pupils, the most important sign is absence of the reflex to light. Next in order comes extreme miosis (pin-hole pupils), and next in importance a variable inequality (one pupil being larger at one time and the other at another time). Irregularity of outline of either or both pupils is significant. Simple inequality of the pupils is less distinctive because met with in other forms of insanity, and occasionally in normal persons. Marked anisotropia is very common in the latest stage of the disease.

In tabic forms of the disorder the knee-jerks are diminished or lost. In all other forms the tendon-reflexes are apt to be enormously exaggerated, so that we get not only extreme knee-jerks, but quadriceps clonus, ankle-clonus, jaw-jerk, jaw-clonus, and extreme wrist- and elbow-jerks. With this spastic condition we observe also considerable rigidity of the muscles, with a tendency in the latest stage to marked contractures. Often in tabic forms, when the knee-jerks are at first lost, they become finally exaggerated. Hence, while the term *tabic* is often used to describe a form of paresis in which we have lost or diminished knee-jerks, together with Argyll-Robertson pupils, this is simply a descriptive designation, and does not necessarily imply that we have a combination of locomotor ataxia with paresis.

As previously stated, one of the chief symptoms of paralytic dementia is a progressive weakening of the muscles in general of the whole body. It is rather an enfeeblement than a paralysis. It is manifested mainly by localized pareses in various muscles or groups of muscles. These are often noted as early symptoms—for instance, in the eyes and face. In fully one-half of the cases we observe, at one time or another, weakness of some of the ocular muscles, not infrequently



giving rise to diplopia or ptosis, rarely nystagmus. A certain amount of ptosis is often seen, and the overaction of the occipitofrontalis in consequence forms a striking picture in many cases. One-sided paresis of the forehead muscle, orbicularis palpebrarum, or lower face is rather common. The muscles about the mouth are particularly often involved, so that marked inequality of the nasolabial fold and of all of the oral movements is encountered. The speech has frequently a nasal tone from one-sided or double palate paralysis. Deviation of the tongue is common. The general strength of the extremities, as measured by dynamometers, is diminished, sometimes on one side more than on the other, presenting the picture of a hemiparesis. The want of equal innervation is sometimes indicated by the attitude of the patient, the inclination of the body to one side or another, backward or forward, sinking of the head on the least, etc. Weakness in the muscles of deglutition leads to difficulty in swallowing. The peculiarity of most of these paralytic phenomena is, in the first place, their mildness of degree, and, in the second, their frequently transitory character (the weakness may be first on one side of the face, then on the other, now about one eye, now in an extremity, etc.).

Nearly every case of general paresis exhibits, at some time in its course, convulsive or apoplecticiform seizures. Usually these critical episodes occur at the height of the disorder or in its final stages, but occasionally they are among the very earliest symptoms. For instance, one case that came under my observation began with a transitory hemiplegia following an apoplecticiform attack. Up to the day before this seizure he had performed his difficult duties as an accountant in a large railroad organization to the perfect satisfaction of his superiors, and none of his family had observed any indication of prodromal symptoms. He died as a typical paralytic a year later. Another case, much the same in many ways, began with general epileptiform convulsions extending over twenty-four hours. The attacks may appear in the form of syncope, or coma, or aphasia. A peculiarity of all of these crises is their transient character, and so even in cases terminating fatally in such attacks often no lesion has been found, their pathogeny has been ascribed to congestive conditions or to circumscribed edemata in various areas of the brain. As a rule, mental failure becomes more apparent after these crises.

Rapid emaciation is usual after the disorder has actually set in,—that is, at the termination of the prodromal period,—but later on, after the climax has been reached and dementia becomes more apparent, patients often gain largely in flesh.

Among the trophic disorders we note especially bed-sores, which appertain mostly to the terminal condition. In some of the cases a true trophic neurosis is the cause, and in others weakened peripheral circulation and uncleanness. A striking fragility of the bones is common in general paresis, which accounts for numerous accidents in asylums, such as fractures of the ribs and other bones, exploited so often in the newspapers as due to the assaults of attendants. I have known a mineral paretic to break all of the small bones of his hand by pounding on a door. Hematoma of the ear is very frequent in paralytic dementia,

and this must be ascribed to trophic changes in the vascular walls, permitting some trivial trauma to cause a rupture in the vessels of the perichondrium. The hair frequently becomes rapidly gray in paresis, and this, too, is doubtless a trophic symptom.

Among other physical symptoms occasionally met with are to be mentioned changes of temperature, alluded to in the chapter on General Symptomatology, intermittent albuminuria, polyneuritis, glycosuria, acetoneuria, polyuria, impotence, and vesical and rectal weakness. Glycosuria is sometimes an early symptom.

As regards mental symptoms, the gradual and progressive failure of memory, and, as a consequence, the progressive depletion of the store



FIG. 116.—Group of paresis. Taken in three excited and melancholic phases (Dr. Kowatz).

of memory-pictures, ideas, idea-associations, and judgment-associations, are the most noteworthy features of the disease. The most complicated conceptions, as well as those acquired latest, are the first to disappear. Abstract ideas, owing to their complexity, are the earliest to go. The patient loses his memory for dates, for the events of to-day and yesterday, and finds difficulty in remembering his appointments and duties. A very early loss of the power of mental computation is notable. With the progress of the malady, even the older memories and concrete ideas vanish by degrees. The patient comes to have no knowledge of time, the place where he is, or of the friends who surround him. The loss of the faculty of judgment is evident at an early period in his failing observation and comprehension of his own symptoms. Ordinarily there is a retardation of the flow of ideas, particularly marked in the melancholic type of the disease. In the excited type there is an acceleration of the flow of thought, which is given a special color by the mental enfeeblement.

There are cases which run their course without delusions, the symptoms then being merely the progressive dementia with advancing physical decay. But in a considerable proportion of paresis delusions are manifested, usually of grandiose character, associated with more or less abnormal excitement (sometimes approaching the manic condition).



tion), and occasionally of melancholy character. The grandiose ideas of male patients are concerned with wealth, power, glory, size, strength, position, possessions, and of female patients with dress, finery, jewels, and children. At an early period these grandiose ideas are not to be distinguished from the similar fancies of many cases of ordinary acute mania. But when the judgment becomes weakened, as it inevitably does, a peculiarly distinctive character is given to the patient's delusions. The grandiose delusions take a magnitude, an enormity, a stupendousness not observed in any other form of insanity. Wealth is counted in decillions of worldfuls of gold. The patient is czar, king, president, queen, God, at the same time. His penis is a mile long, his testicles large diamonds. He will bring the Pacific Ocean over the Andes to make the largest waterfall in the world. He will move the asylum buildings on a rail of gold to Washington. He has thousands of wives, every one of whom bears two hundred children nightly. He bestows on his physicians and nurses royal orders, dukedoms, writes them checks for enormous sums of money, etc. When the mood of the patient is hypochondriacal or melancholic, the delusions retain the same element of enormity despite their unhappy contents. He states that he is impoverished by having lost billions of dollars; he is committed to prison for thousands of years; he weeps because he can not do his duty to the nation which he governs; there is some horrible condition of his bowels which requires the most awful of operations, etc.

There are some cases of general paresis which exhibit alternating phases of melancholic depression and ambitious exaltation, and these are described as paralytic dementia of circular type.

Hallucinations and illusions are frequently observed in general paresis. They have more or less relation to the condition of exaltation or depression present and to the delusions manifested. Auditory hallucinations are the most common. They are noted even in the early periods of the disease, but are generally a part of the maximum period. They are absent in the final stage.

Emotional irritability and changeability are generally evident. The patient laughs or weeps easily, and is often readily angered.

The excesses, sexual and alcoholic, lapses of propriety, etc., are significant of loss of esthetic and ethical sensibility. He indulges himself freely and without morality (though previously moral), drinks immoderately, steals, and squanders his own and others' property. As his character sinks lower and lower he commits all sorts of shameless offenses against decency.

Before passing on to the final stage, we not infrequently encounter, in the course of the disorder, peculiar interludes of recession of all of the symptoms. These are known as remissions. Remissions last from several weeks to several months, as a rule, occasionally for a year or more. There are instances on record of remissions lasting for ten or twelve years, and there is one authentic case in which a remission lasted twenty years, the autopsy showing the characteristic pathological changes of paresis. Very striking at times is the remarkable improvement to be observed in a remission. This may attain to a degree making it almost

impossible to discover any vestige of deviation from the patient's normal mental health. The extraordinary delusions disappear, the maniacal or melancholic mood vanishes, the symptoms of confusion and forgetfulness pass away, and noteworthy intellectual lacunae are filled again. The patient may return to his affairs. It is very rarely that marked physical stigmata of the disorder diminish and give place to normal conditions. After a time the old symptoms of the dread malady reassert themselves and its fatal progress is rebegun.

**Terminal Period.**—As already intimated, there are cases in which



Fig. 112.—A noted actor who recently died of paresis. Taken to show the expression of paralytic dementia in an unusually expressive face (found by Dr. Atwood).

there is merely a progressive enfeeblement of mind and paresis of body from beginning to end, with none of the excited or depressed conditions, delusions, hallucinations, remissions, etc., just described; cases which pass by gradual steps from the prodromal into the terminal period. In the main, however, we have most of these other manifestations interpolated. The final stage is often ushered in by the convulsive or apoplecticiform seizures. This is the stage of more or less complete dementia. We may still note the remains of old grandiose or hypochondriacal delusions in the scarcely comprehensible mumblings of the parietic dement, but usually the mind becomes completely vacuous; the patient speechless, filthy in his habits, bedridden, and more helpless than an infant. He lies in bed, either motionless or restlessly moving his limbs and grinding his

teeth. He can scarcely swallow his food, and often requires to be fed to prevent strangling. He retts and soils himself, and bed-sores and contractures develop. Finally, death by inhalation-pneumonia, septicaemia (from the bed-sores), cystitis, uraemia, intestinal catarrh, or exhaustion steps in to draw the curtain on the distressing picture. Not a few die at an earlier period in an epileptiform or apoplecticiform crisis.

**Duration and Prognosis.**—Paralytic dementia runs its course in three to five years, on an average. There are more cases which terminate under three years than over five, but cases lasting five years are not infrequent. A duration of ten years is among the greatest rarities.

The prognosis is practically always death within a short term of years. The author has never known personally of a case recovering. In our whole literature there are, according to Ziehen, but a dozen cases of recovery on record. It is probably questionable if even these



were genuine cases of paresis, since an error in diagnosis is not at all uncommon.

**Diagnosis.**—The chief disorders which may be confused with paralytic dementia during the various stages of its evolution are neurasthenia, alcoholism, syphilis of the central nervous system, acute mania, epileptic dementia, paranoia, or secondary paranoia with delusions of grandeur, multiple sclerosis, and mental conditions associated with common organic lesions of the brain (tumor, hemorrhage, embolism, thrombosis). In atypical cases the diagnosis is often difficult and sometimes even impossible.

Valuable aids to our methods of diagnosis are found in lumbar puncture and a study of the spinal fluid, to show the presence of a lymphocytosis characteristic of paresis as compared with any of the functional psychoses; also in the Wassermann test and the Noguchi test for syphilis.

As regards neurasthenia, it is only in the prodromal period of general paralysis that differentiation may be difficult. I shall attempt to present in brief, tabular form the distinctive diagnostic points of these two conditions:

GENERAL PARESIS (EARLY PERIOD).	NEURASTHENIA.
Staggered, tremulous, irregular, pin-hole, or unequal pupils.	Large and either active pupils usually.
Diminished, greatly exaggerated, or unequal knee-jerks.	Active and equal tendon-reflexes.
Fibrillary tremor of tongue; jerky, staccato tremor of fingers, face, tongue, occipitofrontals.	Tremor fine and rapid of fingers and eyelids, not jerky; very rarely involving face, almost never the tongue and forehead.
Eision or reduplication of letters, syllables, or words in writing.	Nothing abnormal in the writing.
Sometimes noticeable characteristic defects in speech.	No changes in enunciation.
Usually little or no notice taken by the patient of his symptoms.	Patient pays marked attention to his symptoms.
In some cases a feeling of cheerfulness and well-being out of proportion to the actual disorder present.	Patient apprehensive and alarmed at any symptoms present.
In many cases a vague, hypochondriacal depression with restlessness, not referred to any definite physical cause.	When hypochondriacal, patient's attention fixed on some definite morbid process which he believes to be going on in his system.
Actual evidence generally found of failing memory, defect of intellectual process, weakened judgment, and loss of esthetic and ethical feeling.	No evidence of mental decay or loss of esthetic and ethical feeling.
Occasionally epileptiform or apoplectiform crises.	Nothing of this kind in neurasthenia.
Vertiginous attacks and transitory aphasia of mild degree.	Not present in neurasthenia.
Characteristic serological findings.	Syphilis might exist, but the serology would not be like that of paresis.

In chronic alcoholism we may have presented to us many symptoms, such as tremor, thick speech, mental changes and defects, epileptiform crises, and, where rudimentary polyneuritis is present, list knee-jerks, which may simulate the syndrome of paralytic dementia. The resemblance is sometimes remarkably close, especially in cases of Korsakoff's psychosis. The chief differential point is the great improvement and

often recovery which take place in alcoholic mental disorder on withdrawal of the alcohol. With abstinence the speech becomes normal, the tremor grows less or disappears, the hares-jerks return, epileptiform attacks cease, defects of memory are no longer perceptible. If hallucinations are present, they are more often visual and zoöscopic in alcoholism, while generally auditory in paresis. The delusions of the chronic alcoholic are, as a rule, suspicious and persecutory. It must be remembered that a typical general paresis may, however, develop on the basis of a chronic alcoholism.

Aside from the comparison of neuro-thenia with the prodromal period, probably the mistaking of syphilis of the central nervous system for advanced general paralysis is the most common error in diagnosis. The two disorders have so much in common that their differentiation is often only possible by prolonged observation through the whole course of the disease; and if the parietic dementia should happen to progress as a simple dementia with none of the characteristic episodes, the diagnosis is sometimes quite impossible. The following table will serve to make some of the similar and unlike features of the two maladies apparent:

#### GENERAL PARALYSIS

Paresis of individual groups of cranial nerves at times. Slow in onset and insidious.  
Symptoms of a diffuse general lesion.  
Jerky and ataxic tremor.  
Loss of iris reflex to light, preservation of movement of iris in accommodation (Argyll-Robertson pupil) extremely rare.  
Characteristic clonus and multiplications of letters, syllables or words in writing.  
Peculiar disorder of speech. (G. P. speech.)  
Headaches vague, transitory, and seldom distressing.  
No material changes in the pupils.  
Progressive advance of the disease to a speedily fatal termination, with a possible remission in some instances for a brief period.  
Delusions often expansive, sometimes depressed, characterized by enormous exaggeration in either case.  
Affective state often expansive, sometimes depressed.  
Progressive mental enfeeblement.  
Epileptiform and apoplectic crises in nearly every case, and frequently repeated.  
Antisymphilic remedies usually affect but little the progress of the disease.  
Considerable increase of cells in spinal fluid.

#### CEREBROSPINAL SYPHILIS

Complete paralysis of one or several cranial nerves often. Generally sudden in onset and stable.  
Symptoms of multiple lesions.  
No tremor in ophthalmos.  
Iris often amenable both to light and to accommodation; extreme cases very infrequent.  
If any change in writing at all, due to atrophy or dementia. No resemblance of the changes to those of paresis.  
No speech disorder usually; but, if any, due to organic aphasia of one kind or another. No resemblance to the G. P. speech.  
Headaches extremely severe, constant, and worse at night.  
Optic neuritis occasionally.  
Irregular advance, with many fluctuations in intensity and character of the symptoms, extending over a long period of years, and not necessarily fatal.  
Delusions rarely present.  
Affective state usually depressed or apathetic.  
Incoherence and thought-inhibition.  
Epileptiform and apoplectic crises uncommon, but if they do occur, are generally single, isolated attacks.  
Antisymphilic remedies of marked service.  
Often extraordinary increase of cells in the spinal fluid.



A gummatous meningitis may, however, present a typical general paresis in all its manifestations, and there are cases in which the actual lesions of paresis exist side by side with syphilitic cerebral lesions.

We may have maniacal outbursts in the course of general paresis. Indeed, I have seen paresis begin in a number of instances as an apparent acute mania. During this maniacal state the chief means of differentiation of the two disorders is in the character of the contents of the delusions. Both are exalted and expansive and tend to the same general exaggeration of feelings of power, strength, intellectual and physical abilities, wealth, social station, etc. But the stuporous exaggeration in general paresis is never observed in acute mania. This is a valuable indication. Naturally, if any of the physical signs of paresis are present, the diagnosis is not difficult.

Epileptic dementia, with its slow speech, mental defect, and epileptic seizures, might at times be mistaken for a paralytic dementia, presenting chiefly these symptoms. But the history of long years of epilepsy preceding the psychic degeneration suffices, as a rule, for the diagnosis. It is only when such history is not obtainable that error might arise.

In paranoia itself, and in paranoia secondary to acute mania or melancholia, the expansive or depressed delusions are of a more fixed and much less exaggerated nature. A study of the character of the delusional contents should make differentiation easy.

Multiple sclerosis, with its jerky tremor, exaggerated reflexes, and mental enfeeblement might at times present a syndrome analogous to that of some cases of paralytic dementia. The tremor of multiple sclerosis, however, while also jerky and ataxic, is a marked intention tremor, exhibiting wider and wider excursions the greater the effort to carry on a voluntary movement. The tremor of paresis, on the other hand, shows no such increasing exaggeration on voluntary efforts to use the muscles. In sclerosis, the head is often involved in the tremor; in paresis, never. Nystagmus, so common in sclerosis, is almost never observed in paresis. The dementia of sclerosis, when present, is slight and not especially progressive, and there are no expansive or depressed delusional episodes, such as characterize paralytic dementia.

Focal brain-lesions (tumor, hemorrhage, softening, etc.) with dementia and paralysis may simulate somewhat certain types of general paralysis, but the progressive character of the latter disorder, with its crises and psychotic episodes, should serve to give the condition presented definite outline and character.

The serological tests for syphilis have, as a rule, great corroborative significance. A Wassermann test of the blood and of the spinal fluid is usually positive and the cells in the spinal fluid which normally number three and not above ten, may be from ten upward to many hundreds. A moderate increase, say up to around fifty, is rather in favor of the meta-syphilitic disorder, while a count of hundreds of cells is more common in cerebrospinal syphilis.

**Pathological Anatomy.**—It is usual to describe the pathological condition underlying paralytic dementia in general terms as a diffuse meningo-encephalitis. The gross changes observed at autopsy are as follows:

1. General diminution of weight of the brain.
2. Increased fluid in the subdural space and in the meshes of the arachnoid (external hydrocephalus).
3. Pachymeningitis hemorrhagica interna, with large, fresh, or old hematomata of the dura mater (in about half of the cases).
4. Chronic leptomeningitis (opacity and thickening, with adhesion of the membranes to the cortex).
5. Narrowing of the cortex, with gaping of the fissures.
6. Distention of the ventricles with serum and granulated and thickened ependyma (chronic internal hydrocephalus).
7. Gray degeneration in the centrum ovale, brain-axis, in various columns of the spinal cord, and in some of the spinal roots and peripheral nerves.

The microscopical findings of Alzheimer may be summarized briefly as follows:

1. Proliferation of new capillaries and of the endothelial cells and adventitia.
2. Dilatation and infiltration of adventitial lymph-spaces, with lymphocytes, mast-cells, and plasma-cells, mostly the latter.
3. Degenerative changes in the blood-vessels, especially in the cortex.
4. A peculiar cell-form usually present in the cortex (Nissl's *Alzheimer cell*).
5. Diverse and wide-spread degeneration of the ganglion-cells, but not pathognomonic of paresis.
6. Arrangement of cell-groups in cortex more or less altered.
7. Degeneration of axis-cylinders.
8. Proliferation of glia tissue throughout the cortex, especially about the blood-vessels of the outer cortical layers with noticeable growth of astrocytes.

The whole cortex is more or less affected, but often the changes are more marked in one area than in another. It is usual to find the frontal lobes especially implicated.

**Treatment.**—The strong modern movement against alcohol will be an element in prophylaxis, since so many luetic infections take place under the influence of alcohol, and the increasing public knowledge of the dangers of syphilis together with all the efforts now being made to prevent its spread, should tend to diminish the number of cases of paresis. It is to be hoped that our newer and better methods of treatment of specific infections may also serve to stave off the development of later syphilitic cerebrospinal disorders. We should, therefore, expect to see after a time a smaller percentage of paralytics in our asylum statistics.

In the majority of cases of general paresis commitment to an asylum is necessary, owing to the dangers arising from the patient's excesses. He may squander his property or scandalize his family by his immoral or criminal acts. It is true that cases which present merely the dual symptomatology of increasing physical debility with progressive mental enfeeblement may be, and often are, treated at home. But, on the whole, it is better to act promptly in placing the patient in a place of safety.



While antisyphilitic measures have not as yet demonstrated any certainty of cure in any case of true paresis, energetic treatment should be carried out in the hope at least to induce and prolong a remission.

The serial intravenous injections of salvarsan, or arsenobenzol, etc., may reach the syphilitic germs which we now know to exist in the cerebrospinal tissues in these cases, and at least destroy some of them. Injections should be made at first in small doses three days to a week apart, followed by stronger doses as indicated by the behavior of the patient—twelve—even eighteen to twenty or thirty injections being given, with mercurial inunctions and iodids in the intervals. These arsenical preparations have been used intraspinously also under the impression that the arsenic did not pass into the spinal fluid from the blood, and so reach the nerve tissues; but experience shows that the intravenous injection is just as satisfactory, easier to carry out, and not so dangerous to the patient.

The experiments with injections of tuberculin, nucleinate of soda, and the like have not justified any faith in their being of value.

The treatment of the various symptoms that may arise in the progress of paresis, such as insomnia, excitement, epileptiform and paralytic attacks, dysphagia, retention of urine, bed-sores, etc., is the same as in other psychoses.

## CHAPTER X.

## PARANOIA.

**Synonyms.**—Chronic delusional insanity; Progressive systematized insanity; *Paranoïe*; *Wahnsinn*; *delirium*; *Paranoia*.

**Definition.**—Paranoia may be defined as a progressive psychosis founded on a hereditary basis, characterized by an early hypochondriacal stage, followed by a stage of systematization of delusions of persecution which are later transformed into systematized delusions of grandeur. Though hallucinations, especially of hearing, are often present, the cardinal symptom is the elaborate system of fixed delusions.

The hypochondriacal stage is called by Régis "the period of analytic concentration"; the second stage, "the period of delusive explication"; the final stage, "the period of transformation of personality."

**Varieties of Paranoia.**—There is one typical form of paranoia to which the main portion of this chapter will be devoted, because it is the type which will be most readily recognized by the student and general practitioner. But there are incomplete or immature forms and atypical variations, which the special student of morbid psychology learns in the course of time to distinguish. Thus, many of those eccentric or queer individuals whom we call "cranks" are rudimentary or undeveloped cases of paranoia. Some idea of the varieties of paranoia noted by authorities may be gathered from the attempts at classification by different writers. For instance, French and Italian authors are inclined to divide paranoia into two great groups—viz., (1) degenerative, with original and late subvarieties, according to the period of life at which the insanity develops; (2) psychoneurotic, with primary and secondary subvarieties, according to whether it develops primarily or secondarily to another insanity.

Ziehen classifies paranoia into two great groups, according to the predominance of either delusions or hallucinations—where hallucinations are the most prominent symptom, he terms the psychosis *paranoia hallucinatoria*; where delusions are prominent, he denominates it *paranoia simplex*. Either form may be acute or chronic. Hence he makes four chief types: (1) *Paranoia hallucinatoria acuta*; (2) *Paranoia hallucinatoria chronica*; (3) *Paranoia simplex acuta*; (4) *Paranoia simplex chronica*.

This last form is the name given by Ziehen to the complete typical form of paranoia which is described in this chapter, and which he describes as having four stages (prodromal, persecutory, expansive, and pseudodelusory). Ziehen also specifies several varieties of acute hallucinatory paranoia—viz., the fleeting-idea form, the stuporous, the incoherent, the excited, and the depressive forms.

Krafft-Ebing makes two great divisions—original paranoia, appar-



ing in early childhood or before puberty, and acquired (*facile*) paranoia, appearing between the ages of puberty and sixty years. The latter class he subdivides as follows:

(A) Paranoia persecutoria: (1) the typical form; (2) solitaire (*paranoia sexualis*); (3) *paranoia querulans*.

(B) Paranoia expansiva: (1) *paranoia inventoria* and *reformatoria*; (2) *paranoia religiosa*; (3) *paranoia erotica*.

**Etiology.**—Heredity is a more important etiological factor in paranoia than in any other form of insanity. Krafft-Ebing states that he has never seen a case without hereditary taint. Tausk and Riva found in their cases of paranoia 77 per cent. of heredity and 9.4 per cent. of infantile cerebral disorders, while in the remaining 14 per cent. hereditary factors could not be ascertained, but were not, of course, excluded. It is more common in females than in males. It affects by preference individuals who are even from childhood peculiar, morbid, shy, irritable, mistrustful, and misanthropic. It is very common to find, in cases of paranoia, some of the curious stigmata hereditatis described in the chapter on Etiology, such as cranial or facial asymmetry, malformations of the ear or palate, etc.

**Symptomatology.**—We will examine the symptoms of the different stages in the order of their development. In the *prodomal* period, the hypochondriacal stage or period of subjective analysis, as it has been variously termed, which may have its inception in early childhood, the patient is morbidly shy, peculiar, eccentric, avoids the companionship of others, and is prone to withdraw himself into the solitude of his own thoughts. The physiological commotion of puberty and adolescence, with its inflow into consciousness of innumerable new sensations, its flood of new instincts, powers, ambitions, and ideas, tends to intensify the morbid peculiarities already evident. The patient notes his own peculiarities of conduct, and begins to recognize the singularity of many of the somesthetic sensations which come to him—sensations which at this time might well be considered more or less neurosthenic in character: paresthesias of the head, trunk, viscera, and limbs; pains in various parts of the body, tinnitus aurium, sparks and dots before the eyes, and the like. The unnaturalness of these sensations leads to his spending much time in contemplation of them, so that a hypochondriacal complexion is given to his thoughts. To these physical sensibilities are now added a consciousness of difficulty in the concentration of his thoughts; a difficulty in the proper control of the direction and subject matter of his thoughts. He becomes extremely introspective, and, the more he studies the somesthetic sensations brought to his attention, the more he contemplates the phenomena of the uncontrollability of his thoughts, of their rising unbidden from his subliminal consciousness, of the unrestrained constellation of his presentations, the more is he inclined to search for some cause of his morbid condition. At first, like an ordinary hypochondriac, he investigates himself to find a solution of the problem, and, failing in that, he extends the region of his observation to his environment, seeking there the reason of his strange feelings, general disquietude, and morbid stream of thought. He be-

comes wholly preoccupied with himself. He can not employ himself, either physically or mentally, as he should. He fails in his duties—in everything he undertakes. People seem strange to him in their conduct and in what they say. He grows suspicious and distrustful of everything and everybody. What is done and said by others appears to have some significant relation to himself. People alter in their conduct toward him, look at him curiously, smile sarcastically when he passes, wink at or make signs to one another when he is near; make observations among themselves which, overheard by him, are construed as having a double meaning, as being derogatory to him, reflecting on his character. The more he studies the extraordinary condition of affairs, the more gloomy, solitary, and self-absorbed he becomes. Naturally, the growing alteration in himself really does provoke the notice of others—a fact which tends to intensify his ever-increasing suspiciousness of concealed animosity among those with whom he comes in contact. Many things in his past life rise up in his memory to find a new interpretation in the light of his present general distrust. His physical sensations have become more marked, have taken on a new character, have altered from paresthesias to illusions, and even hallucinations, of general or special sensibility. He feels peculiar general sensations, shooting pains, sudden prickings in his skin. Unusual and unpleasant odors or tastes harass him. Extraordinary sensations flow into consciousness from his genital organs. Much more serious and remarkable, however, are the peculiar changes in his auditory perceptions. At first these are usually confused noises, or roaring and tinkling sounds, with the gradual perversion of sounds and words heard into illusions colored by the suspicious contents of the patient's consciousness; later, actual hallucinations of hearing, which become a fixed and permanent feature of his malady.

The patient now enters into the second or *persecutory period of* mania, the period of delusional explanation of his troubles. He has arrived at what he conceives to be a logical result of his reasonings, a rational explanation of the distress and affliction he has undergone. Everything he has suffered has been due to the machinations of unknown enemies. The delusions of persecution are at first somewhat confused in character. No particular individual or group of individuals is thus far responsible for the inflictions. It is simply some unknown persons who take pains to manifest ill-will or malevolence toward him. "They" talk against him, call him names, attempt to poison him with gases or by tampering with his food, and try to injure him with electric shocks or by throwing corrosive substances at him. Since wherever the patient may be, wherever he may go, the voices, shocks, poisons, etc., seem to pursue him, he comes to think that no single person could manage so vast a conspiracy. It must be some large aggregation of persons who are concerned in the effort to humiliate, cripple, or destroy him; an aggregation bound together by ties of secrecy, and able to permeate all classes of society. What could such body be but a secret society, an order of Masons or Odd Fellows; some religious or political brotherhood—the Jesuits, Catholics, Protestants, anarchists, or police,



Perhaps some one individual is at the head of the band of plotters, some arch-conspirator, but the work is done by innumerable *códes*, who employ all manner of means and apparatus to accomplish his ruin. This system of persecutory ideas is built up in the most elaborate way, and the more educated the individual suffering from paranoia, the more wonderful the organization and adjustment of the various parts of the delusional system. The persecutory delusions of other forms of psychoses, such as toxic insanity, senile dementia, and melancholia, may have a certain interest and fixity, but those of the paranoiac are woven together like a romance. The relation of the former to the latter is that of the brief sketch to the serial novel. The telephone, the phonograph, telepathy, hypnotism, and other and more mysterious apparatus and phenomena are brought into service by the relentless league. I do not know the origin of Du Maurier's conception of his novel, "Peter Ibbetson," but I suspect that many of its unique features, especially that of "dressing true," were suggested by conversations with some well-educated paranoiac in a lunatic asylum.

Many patients seek in a most elaborate way to explain one peculiarity of their auditory hallucinations—viz., the fact that their thoughts are read off by the voice or voices simultaneously with the appearance of the thoughts in consciousness. This adds naturally a new terror to the persecution, for the ability of the conspirators to read off and taunt the patient with his own most secret thoughts is a particularly refined species of devilry, as well as evidence of the extraordinary psychological power of his tormentors. The voice which speaks his thoughts, or answers his thoughts before he can himself utter them, may be referred to the external world or to some part of his own body. This phenomenon has been variously termed echoing of the thoughts, motor representation of articulation, and verbal psychomotor hallucination. It depends upon the close relation existing from earliest infancy between the auditory word-center and the motor speech-center. Any irritation of this auditory area is immediately, synchronously, irradiated to the motor speech-center. However slight this stimulation of the speech-muscles, recurrent sensations of movement in them are carried back to the brain, giving rise to the hallucinations of internal hearing.

The patient is driven by his delusions to make complaints to the



Fig. 222.—Young paranoiac with homicidal tendencies at period of passing from persecutory into grandiose stage (Dr. Akenside).

police, to judges, or to the governor of the State, the President, or other government or judicial authorities. Not infrequently he attempts, himself, to wreak vengeance upon one or more of his imaginary enemies. Attempts at homicide are, therefore, common in these cases. The writer had in his charge at the Poughkeepsie Asylum, for some years, Ernest Dubourg, a persecutory paranoiac, who, many years ago, ran through Fourteenth Street, New York, stabbing women right and left with a pair of compasses. More often they seek to escape from their enemies by constant change of residence.

The third stage, the *expansive period*, or the period of transformation of personality, is often induced by the patient's attempt at a logical explanation of the cause of the persecution. Since he has so many enemies, and every man's hand is against him, it must be due to his importance. He either resembles some distinguished personage or he



Fig. 105.—Duchenne's "Mary Appleton's head" (see text).

is of royal or god-like descent. The transformation may be suddenly induced by a hallucination revealing to him his high estate. The contents of these delusions of grandeur may be religious, political, erotic, jealous, and so on. For instance, the delusion of being a prophet or a second Messiah is very common (*paranoia religiosa*). The delusion of being a great discoverer or inventor is frequently met with (*paranoia inventiva*). Another common delusion is that of being a great social reformer (*paranoia reformatoria*). A peculiar form is *paranoia erotica*, in which a person imagines him- or herself to be beloved by some one of superior station. It is a romantic, platonic love in which the patient indulges. He has communications with the object of his delusions, imaginary conversations,

through the medium of hallucinations. A good example of this form was that of Dougherty, who followed Mary Appleton all over the country, and was finally sent to an asylum because of his threats to kill any one who interfered with his attempts to gain a personal interview with the famous actress. Measurements which I made of his head showed a pathological excess in the height of the skull.<sup>1</sup> After his commitment to an asylum he shot one of the physicians who had him in charge. Another interesting variety of *paranoia* is that observed in the litigiousists (*paranoia querulans*), who occasionally distinguish themselves by their lifelong involvement in legal processes (due to an overwhelming egotism, which leads to a continual zealous effort to set themselves right, despite the advice of friends, and the wasting of their property, after the

<sup>1</sup> "Tumid Form of Nervous Disease," by M. Allen Starr, New York, 1890. Article on "Paranoia," by F. Peterson, page 299.



loss of some possibly trivial lawsuit). Pretenders to thrones, self-styled kings, presidents, princes, and so on, are often noted among paranoiacs who have reached this third stage of evolution. Quite commonly persecutory ideas still remain in the minds of these patients in association with the delusions of grandeur.

Each of these periods of development may last for several years, the disorder may undergo arrest at any period, and there may be variations in the degree of development of any stage; so that we constantly meet with atypical forms of paranoia. An excellent condition of memory, judgment, and intellect in all other directions save in those related to the single cluster of delusions may exist. Years ago these cases were designated as *monomaniacs*, because of the apparent facility of the patient outside of the limited number of fixed ideas. Many paranoiacs have distinguished themselves in sacred and profane history, and even in literature. There have been many of these false prophets who have come to herald a new religion—Mahomet, Swedenborg, Joanna Southcott, John of Leyden, John Thoms of Canterbury, and some say Jeanne d'Arc. We have had them even in the United States within a few years—the leaders exploited by the press. Among political reformers we had John Brown and Guiteau. A famous paranoiac immortalized himself in his autobiography—Benvenuto Cellini.

I have in my possession a beautifully written manuscript—the autobiography of a paranoiac. He was so dangerously insane that he spent much of his life in the asylum in which he wrote this valuable work.<sup>1</sup> The volume, bound by himself, is entitled "The Piling of Topknot," which is significant of the sufferings he had undergone in his unhappy life. I believe no better idea of the typical form of paranoia can be obtained than by a careful reading of the history of this case as given by the person himself. It is a graphic picture of the steady evolution of the malady—a remarkable self-dissection of the soul's anatomy. Before presenting the extracts from his autobiography, I shall make a few transcripts from his *nevrin* history.

He was thirty years of age at the time of admission; single; a farm-laborer by occupation. He was not a church-member, had a common-school education, and was a native of the United States. Hereditary predisposition was not acknowledged. His mother, who accompanied him to the hospital, stated that he had always been delicate in his physical constitution, and given to despondency. Since the age of twenty he had done little or nothing, because of ill health. A year previous to his commitment to the hospital as a lunatic he shot himself in the forehead in an ineffectual attempt at suicide. Later, he developed delusions that the people of the village were acting upon him by suggestion, spoke disparagingly of him, and were conspirators against his peace. During the whole of his sojourn in the hospital he had hallucinations of hearing, and in the earlier period of his stay had delusions of persecution. Toward the end of his seven years of hospital life he gradually developed, in addition, delusions of grandeur. Although he

<sup>1</sup> "Extracts from the Autobiography of a Paranoiac," edited by Frederick Pyramus, "Am. Jour. of Psychology," January, 1899.

had occasional lapses of self-control, manifested by the breaking of window-glass or the tearing of clothing, he was for the greater portion of the time sufficiently self-possessed to restrain whatever violent or destructive inclinations he may have had, and was permitted to go out alone upon the large grounds of the asylum whenever he wished, and to wander about the woods at will.

It was during the last two years of his stay at the asylum, while still the victim of constant auditory hallucinations, and of mingled delusions of persecution, un-even agency, and grandeur, that he wrote the volume of four hundred manuscript pages with the extraordinary title of "The Piling of Tophet," this title being founded upon Isaiah xxx, 33. The book itself is a deeper history of his life and mental evolution than any but himself could furnish. It is remarkable for its excellent literary style and for its keen reasoning and psychological analysis of his own disordered mind. In it he dissects his hallucinations and delusions like a skilled anatomist. It is as fascinating as a novel. Every page has its value as an index of the condition of his mind from childhood to the last years of his confinement in the asylum; and the story is told with a directness and simplicity that marks truth upon every statement and lends it such charm as pertains to all works which portray life with the utmost fidelity. In his preface and introduction he makes a diagnosis of his own disease.

Our author, as has already been stated, was not a church-member, and in his book he describes his early religious life and his subsequent beliefs as they developed. His father was a Universalist and his mother a non-professor of religion, although she did attend the Methodist church. During his boyhood he attended the Sunday-school regularly, and at one time the Episcopal church; but his attendance upon divine service ceased in early youth. Both parents were honest, conscientious, and highly respected in the community. They were first cousins. The mother was healthy in mind and body, but the father is reported to have been exceedingly eccentric, possibly insane. From what I subsequently learned regarding him, he also was something of a paranoiac. They strove to bring up their children carefully and to educate them as well as possible.

His father died when the patient was twelve years of age. Up to the age of thirteen he attended a country school both winter and summer, but after that his farm-work permitted him only winter schooling. Still, he evidently had unusual talents and aptitudes, and we find him later studying by himself, in the original, many of the classic Latin authors; and among his favorite companions were the works of Boethius, Laetantius, Josephus, and the Bible. His literary style and modes of thought are in themselves an evidence of more than ordinary attainments in rhetoric, philosophy, and logic.

The matter of heredity in his case was not sifted thoroughly upon his admission to the asylum, nor have I since been able to gather much material relative to this factor in his evolution. But one important element of this nature is described in his book—an element not only hereditary in its character, but for a long time part of his environment,



and undoubtedly an influence modifying his mental condition both before and after his birth. I allude to a great-uncle, a brother of his grandmother on his mother's side, who was himself a paranoiac, and who lived upon the farm in intimate companionship with our patient until the latter was twenty-three years old.

As we read on we see, from the author's account of himself, how heredity and environment gradually moulded his physical and mental characters. A shy, timid, delicate child; clever intellectually; given to oddities of speech and conduct; inclined to solitary musing, rarely sharing the sports or games of other boys—in him were slowly evolved marked eccentricity of demeanor, a disposition to shun his fellows, a misinterpretation of their looks and actions as regarded himself, a morbid egotism, a consciousness of a gulf between himself and ordinary men, with deep depression, outbursts of passion, an inclination to homicide restrained but feebly by his weakened will, and delusions of persecution. No doubt the derogatory remarks he fancied expressed about him in the stores were the first harbingers of auditory hallucinations. Later, he had murder in his thoughts, through the morbid humiliation he felt at the imaginary insults from others. No doubt, as his conduct grew more and more strange, he did attract attention among his fellow-men, and this, unfortunately, would but feed the flame of his pathological self-consciousness.

We follow his history from infancy through childhood and youth to manhood, and observe how, slowly but surely, the hereditary seed sown in degenerative soil took root and flourished. His peculiar auditory acuteness, with his morbid shyness, soon gave rise to illusions of hearing, and these again were transformed into hallucinations, as is evident if the thread of the narrative is carefully followed. The curious foundation of his hallucinations he well illustrates and understands. An idea arises in his own mind of what people would say in discussing him, and immediately consciousness in the auditory area projects the idea in spoken words into the environment. He noted this peculiarity of his own thoughts being repeated to him by the voices about him, yet he could not correct the delusions to which they gave origin, but interpreted the matter with the reason and judgment of an insane mind. He naturally had the delusion, founded upon his hallucinations, that people were persecuting him, but upon this now grew another delusion. He began to believe that they could read and repeat his thoughts; that there was some magnetic means by which his tormentors could draw off his thoughts; that other wills could act upon his body, dominating his own will and causing him to do things he had no desire or intention of doing.

It was about this time that he was removed to the asylum. Several chapters of his book are devoted to a description of his life there, his religious beliefs, illusions, and hallucinations. A short time previous to his departure for the asylum he began to read much in the Bible, and, as he says, noted passages which seemed to have a special bearing as regarded himself. There were several coincidences of this kind, and he looked upon them at first as surely coincidences, but in time the

resemblance became so strongly marked, to his disordered intelligence, that he came to look upon whole chapters of the Bible as referring to himself. From this the step was not a great one to the delusion of being a prophet. In reading we find that our author had several incentives for writing this book. It contains the autobiography of a new prophet, as well as the revelation of a new religion. From his standpoint, as a man in whose destiny are wrapped up the destinies of the world, he tells posterity of the fortunes and trials he has passed through as an atonement for the sins of the earth; how he was mocked and scoffed at, his brain acted upon by magnetic agency, and himself imprisoned in a lunatic asylum for years. Hence the title of his book, "The Piling of Tophet." But behind this insane egotism there shines at times some faint glimmer of the truth, so that he frequently speaks of himself in the terms used by his fellows, as insane, a lunatic, a monomaniac, as having hallucinations; and he thinks the opinions of his friends, relatives, and physicians of sufficient worth to merit considerable argument in his book. He knows what insanity is; he recognizes it in his asylum associates. He could at times "see the man he ought to have become rising up like a shadowy phantom in judgment on the wreck he really was." But this occasional consciousness of their disordered mental condition is by no means infrequent in the insane.

Shortly after writing his autobiography he was removed to a county asylum, where he remained, without change in his mental condition, for several years, when his friends took him out to live with them. He died a religious paranoiac in 1886. He did not become completely imbecile, as such cases often do; we did he write any further articles, so far as I am aware. Doubtless the indifference with which the world received the propaganda of the new prophet caused his philosophical withdrawal from active warfare in the fields of reform and theology.

In the preface he defines the scope of the book as follows:

"This work is given to the public as a lunatic's defense of his position. Every effort I have made hitherto to come to an understanding with my fellow-men, on things which I see to proceed from them, and which give my life its whole shape, has drawn out nothing more than blank denials of all knowledge of the things I spoke of. Now, it is impossible for me to reduce my thoughts to the bounds which others have been willing to concede. The object of this little autobiography is to show the form and consistency of the thought that is in my mind.

"I present my evidence to the tribunals of last resort, the public and the press, and ask them to try the case and render their verdict. Have I a right to my thought, or have I not? If not, where am I deluded? If I have, why is not mine the true thought for all men?"

A paragraph from the introduction further reveals the object of his confessions:

"A person is supposed to have a reason for what he does, and I might consider it incumbent upon me to tell the motives which actuate me in thus entering upon the work of this scribe under circumstances so peculiar. Is there anything I have to tell that might not as well and



more safely be left untold? It is a question which I do not have to consider and decide to-day, for I have been long inspired with the conviction, the consciousness, that I have something to tell that it would be worth the world's while to hear."

In another introductory paragraph he makes an excellent diagnosis of his mental infirmity. Addressing his reader, he says:

"I did not tell you that I am a patient in an asylum. I am to take it for granted at the outset that my prospective reader knows nothing of my character, condition, or circumstances beyond what I tell him. I am here as an insane patient. I have been here over five years. Being an insane man, it will be nothing unexpected that I should, in giving those reports of my fortunes, narrate incidents and particulars partaking more or less of the marvellous or preternatural. I am not only a lunatic, but one of the class of lunatics having a controversy with the world in general; in other words, possessed with a monomania, or crazy one-sidedly or on a single subject."

In the hospital record presented above, nothing is alluded to as heredity in this case, and but little stated concerning his mental condition in early youth. These deficiencies are, to a great extent, supplied in the autobiography. I shall permit our author first to describe his appearance in this world, in a cyanotic condition, and the characteristics of his childhood and early youth, and subsequently the hereditary influence in his destiny:

"It is said that I was entirely black when I was ushered into the world, and that for I forget how long a period of time I did nothing but give vent to heart-saddening wails. Was I lamenting the gift of light, on this morning of what was to become a most-burdened existence?"

"I was a weakly infant. I came near dying of the whooping-cough, and it was always asserted, by those who knew, that I owed my life to the uniring exertions of a poor woman who lived a neighbor, who busied herself all night with me, dipping me at intervals into a tub of warm water. My half-sister had it at the same time and died."

"It will be of use to give an idea of my nature and disposition in my tender years. I was always a shy, retiring child; not disposed to make free with strangers; not much given to prattle—in fact, one of the sad and silent sort from the first. I can remember some peculiar sensations which used to weigh on my mind, which go to show that the foundation of my mind-life was but imperfect from the first. I used to be troubled with very strange feelings when I was waking out of sleep, especially if I had been taking a nap in the day-time. It used to seem to me that I was floating in the air, and I often thought to myself: 'Why, how queer I have been feeling!' It was as if I filled the whole room, way up to the ceiling. I was told by others that I sometimes raised myself up in bed after getting to sleep and made an outcry, 'Oh, don't! Oh, don't!' seeming to be in great distress; but the strange part of it is that I could remember nothing about it. I do not think that I ever remembered even their waking me, or finding them at my bedside. I only had their word for it next day."

"As far as I can go back, I remember having at times, but not fre-

quently, impressions which must be identical with what I have lately heard others speak of as 'double memory.' The feeling would all at once creep over me that the very thing I was present with, my ideas and perceptions at that time, had happened to me once before in just the same sequence and arrangement. I have heard this explained as due to a lack of simultaneity in the action of the two lobes of the brain, the tardy one remembering what had already passed through the other. My own theory was different, leaving the organ acting out of consideration. I only went so far as to look at it as a mistaken quality in the perception—an erroneous attaching of the nature of the act of remembering to what was really the act of thinking in the present.

"I was very early in life an observer of my own mental peculiarities, to a degree which I think must be a very rare exception. I often used to be sensible of an unsatisfactoriness in my consciousness of what surrounded me. I used to ask myself, 'Why is it that while I see and hear and feel everything perfectly, it nevertheless does not seem real to me? It is as if I were in danger of forgetting myself and the place where I am!' I often wondered even how I kept the run of things as well as I did. I always found myself holding on to the orderly and proper connection of my acts, and yet from my feelings I could not have answered for my doing so. I can remember sitting at my desk in school, when a small boy, and dwelling with melancholy on this dimness in my perception of existence, and wondering how it was with others in this respect. I wondered to myself if life, as ordinarily bestowed, included this deficiency.

"I showed in my tastes and behavior a harmony with the internal composition of my mind. I was never given to the active sports which the common run of boys take so much delight in.

"The simple fact is that I had a languid nervous development, and from the necessity of my organization could not have much capacity or relish for sports of agility.

"If I could compound a boy of my own I should try to improve on the model I remember to have exhibited in myself.

"It is not true that I was regarded or treated as strange or deficient in my wits. Such an idea would look misplaced to those who knew me and consorted with me in those days. These differences are perhaps more evident to myself than they ever were to the greater part of my acquaintances. I brooded on this side of my character at a later period, and I no doubt remain liable to give greater prominence to disparaging traits than some impartial observers would justify me in doing.

"As a general rule, my harmless and peaceable disposition kept me out of squabbles with my schoolmates. If I was approached in an aggressive way, I met it with absolute non-resistance, which in my case had the disarming effect which is attributed to it by pious moralists.

"If we change the scene from the playground to the schoolroom, we shall find that I attained a distinction of my own, apart from the average, and more to my advantage there. I was always a favorite with my teachers. I never gave them any trouble, and took to my studies with a willing relish that could not but be pleasing to them. I learned to read before I went to school; in fact, like an old asylum acquaintance, Mr. M., inventor and infidel monomaniac, I can almost say that I can't remember when I could not read.

"I was frequently singled out for complimentary remarks on my



proficiency in my studies. I gave evidence of some talents of a higher kind—could draw, for instance, better than any boy in the school.

"One of the most marked weaknesses of my character, as a child, was my susceptibility to being teased.

"After having pondered some on the traits of the human animal in this particular, I have come to the conclusion that there is no further explanation needed than that the impression made on the teaser by the teenable is such as to naturally prompt the acts constituting the teasing, as the sense of burning makes us shrink, and an aroma suggestive of a fine flavor tempts us to bite. I feel convinced that the liability to be teased rests on a principle that has a mighty influence in the motions of the soul of humanity.

"My misdeeds, as a child, were rarely prompted by a love of mischief or the result of headlong thoughtlessness.

"I had a well-defined idea of the nature of sin, and I used frequently at night to recall the events of the day, and reflect on instances in which I had transgressed and given way to ill-humor, and form resolutions to try and do better. From some of the most flagrant of the sins and improprieties to which small and larger boys are prone I was entirely free.

"My early training can not be said to have been a profoundly religious one. My mind was neither imbued with ineradicable prejudices nor prepared for reaction to the other extreme by excessively rigid sectarian drilling and formalism.

"I worked steadily upon the farm, though with moderation, at such kinds of work as I seemed to be equal to. The heavier kinds of work, such as plowing and wagoning, as also the marketing of the produce, were attended to by my great-uncle.

"It is a somewhat delicate subject to manage to my satisfaction this that I am about to enter upon, but it demands candid and impartial treatment, because the events that followed in later years can not be rightly understood without it. It is impossible for me to give a veracious sketch of my soul-life during this period without dwelling quite minutely on the characteristics of my great-uncle. He was a man who had roughed it a good deal in the world, had been at one time in his life a live-color in Florida. How his temper and disposition may have been at an earlier period I can not say—I only remember him as a man possessed of the belief that a certain young man living on an adjoining farm had the power to torture him at his pleasure, both by bothering his brains and inflicting physical pain; which power he made use of to such good effect that the poor victim was almost constantly kept busy holding him at bay by means of runnings of the most fierce and vigorous description. While at work with the horses in the fields, and when driving, he would intermix his commands to the animals with savage execrations of the troubler of his peace. The unfortunate man was troubled, at certain seasons of the year especially, with sore feet, and at such times his imprecations against the offender would fairly rise to yells, and were almost blood-curdling in their intense ferocity. Thus it went on day and night. He slept in a small room in one of the outbuildings, and often he could be heard at a great distance off shouting out threats, sometimes throwing abusive fool-jacks against the boarded side of the building where he lodged to put in the interjection points.

"It may be imagined that a boy of a reserved and sensitive disposition, as I was, could not assimilate very well with such a character as this. I was always distant in my intercourse with him, and a feeling of aversion for his habits of savagery led me to avoid coming in contact with him more than was rendered necessary by our joint labors on the farm.

"As the years passed on, and I continued to live in the presence of my uncle's fierce demonstrations of hostility against the invisible destroyer of his conduct, my tolerance for his conduct immensely gave way. I had now reached the age of eighteen or nineteen; was a tall, slender youth, not strong either in nerve or muscle.

"The exhibition of his ruling passion called up more and more determined feelings of antagonism in my breast.

"Before I knew it I had gone a criminal length in my resentful feeling. I came at last to feel that a person of such a thoroughly savage character did not deserve more indulgence than a mad dog. My position from that time was one of contingent murder. Alas! that I should have been content to let such a state of things last a single day. The frightful danger of my situation ought to have been sufficient to spur me to sacrifice everything to escape from it. But I was in chains, the chains of apathy, impotence, and incapacity, and I could only stay where I was and fume against the object of my detestation.

"I must always regard it as one of the most unfortunate things in my unfortunate career that I should have been placed in contact with this much to be commiserated sufferer at such a time of life. It was not the man himself that I hated. When my judgment could act without impediment, I saw that his unpleasant behavior was entirely the phenomena presented by his never-ending war against what was, in his eyes, the most wicked and cruel of persecutions. I could then pity him and dismiss all malicious thoughts."

This antipathy led to a change in the residence of our author. He felt that he must be separated from his uncle, and, accordingly, he removed to a town at some distance from the farm. It is curious that he never speaks of his uncle as insane, and it is probable that both his mother and him-self and other relatives regarded his persecutory delusions as merely evidence of eccentricity. Soon after removing to town he had some pulmonary difficulty, and he speaks at some length of this as follows:

"In the depressed state of my nerves I imagined myself much worse than I really was, and, like many others in the same condition, I felt as if I was liable to sink away and die at any time. My disease was accompanied with periodical accessions of fever, and in the fictitious strength of excitement given by this my mind seemed to gain an abnormal activity. It was at this time that I first received a revelation on the mysteries of the human soul that had an all-dominant effect on my destinies and the turn of my thoughts ever after. I now learned what had always been to me a hidden mystery—what was the meaning of strength of will and strength of intellect. Before, I had ever lived enshrouded in mists and clouds. In that transitory strength given by the fever coursing through my veins, I now saw the man I ought to have become rising up like a shadowy phantom in judgment on the



week which I really was . . . . My agitation was so great that my mother and the neighbors seemed to fear that I was going crazy. I felt that I had been crazy for a long while and had just recovered reason. It was a fact. But I was constrained to lock up my remorseful agony in my own breast."

We have seen that our patient was throughout his early youth morbidly subjective, and his hypochondriasis increased with years. He had now attained the age of twenty-three; we shall let him describe his mental condition and habits of life at this time. In this description we shall see the gradual growth of persecutory ideas upon a favorable soil:

"My strength and endurance were not sufficient for manual labor, and I did not feel confidence enough in the clearness and energy of my mind to justify me in making application for any post where head-work would have been demanded, or for which ready presence of mind or a good address would have been required. But it was the unpleasantness felt on contact with my fellow-men that operated more strongly than anything else in binding me down to the course of life to which I devoted myself. I felt my deficiencies most keenly every time I met a human being face to face. . . . I could not do otherwise than shun what was so galling to my sensibility, while appearing to conform to no desirable end. . . . But I am going to show that I still remained exposed to very great dangers, and it is as true as it was before that I shunned the only means of averting the calamities threatening me, no doubt of necessity at this stage, and in obedience to the eternal decree that every tree shall spread out and develop in accordance with the qualities given to it 'before it was in the ground.' I did not like the constraint imposed upon me by the presence of man. I did like the freedom of solitude. I strongly disliked many things I noticed in the manner and words of some I met, and there was nothing to prevent this dislike from occasionally being absorbed into my solitary musings, to find its final resolution in the passion of indignation in its various degrees of intensity as the case might be. I have spoken before of my defective means of defense against 'bawling' or 'mocking' for the purpose of troubling. I was always terribly alert and sensitive to all kinds of 'snubs' and sneers, and oblique remarks in general, on their proficiency in which some people pride themselves so much. . . . I was also disagreeably impressed by the ways of some who showed a disposition to turn their attention to myself, instead of confining themselves to the subject I was presenting to them.

"I was being carried into a state of secret enmity to mankind in general by the prevailing tenor of my troubling meditations, and there was no corrective present.

"But all recoiled a hue from a yearning for what was worthy in life, paired with a mournful sense of its hopeless absence. Whatever wrong turns I may in my weakness have been betrayed into, it is impossible that I should look upon my then existing frame of mind as a whole with repentant feelings. As well condemn righteousness and holiness itself!

"When I admit that I occasionally was overcome with an irruption of hard feelings toward wrong-doing men, it will, of course, not be understood that I was habitually morose and spiteful in temper.

Nothing could be further from the truth. What commotion there was was mostly internal, rarely reaching the surface in visible ebullitions.

I occupied myself with the trifling labors of my garden, dwelling with interest and pleasure on the progress of my crops and flowers, and every now and then took a ramble over to the woods lying to the south, which were a favorite place of resort to me all the while I lived there. There I became and moralized, explored the recesses of the woods, enjoyed the calm quiet of nature, and grieved over my hapless condition, wondering what it was to come to.

There were some little things that happened to me the first year after I left the farm which became, as it were, a kind of sample of what I must continue to expect, and the memory of which had more influence over my action in after time than I was aware of myself, no doubt. . . . When I was around the city, thinking I might get employment I called on one of my old acquaintances, who was then in a store. I talked with him a few minutes at that time. I called again a short time after, when I was told by the proprietor that the gentleman I had called to see was not in. There were a number of men present in the store,—salesmen,—and it became apparent to me that they were trying to exhibit an offensive demeanor toward me, or perhaps it would be as true to say that they were moved to make a derisive demonstration against me. At all events, all, with perhaps the exception of the proprietor, stood with countenances of countenance, which was perhaps lighter, until I retired. . . . I found it hard to consign this to forgetfulness. At first it lay dormant, but it would come up, and I must confess I had hard feelings, even revengeful feelings, toward the actors. Another thing happened the same fall. I went to a store, and, standing at the counter, was noticed by one of the clerks,—an Irishman,—who came to me and said, 'I always wait on the little boys first,' and, as I took no notice of the remark, seemed so determined his words should not be lost on me that he repeated them, with the addition, 'like you.' As before, it produced no immediate effect, but it afterward rose and rankled in my memory, and I was not able to keep clear of imagining vindictive things. In fact, to tell the truth, in both cases I felt that blood would have been sweet to me.

My mode of thinking on these incidents no doubt had in it much of the character of insanity. . . . The effect was that I got settled down into the fixed idea that contact with the thoughtless, evil world, in my state of body and mind, would impose upon me the necessity of committing crime in vindication of my honor. . . . I let these bloody memories tinge my whole mind, and all its anticipations and resolutions for the future. . . . 'I see,' I said to myself, in substance, 'that these galling collisions are the natural penalties of being imperfect.'

It may be as well, for the prevention of misconceptions, to say that I never took one step toward putting any design thence arising into execution. I had no designs. I never armed myself, or, in fact, went any further than to rehearse the drama of revenge in my own mind. The pistol I bought was one which I would not have trusted for a moment to carry for the purpose of self-defense. . . . Nevertheless, the events on the farm show that my wickedness was not altogether of a mimic kind, and I will not attempt to escape righteous judgment.



"I used to make many resolutions about regularity in habits of eating, which I found myself powerless to keep. A sense of depression and vacuity would come over me, aggravated by my solitary, monotonous life, I presume, and often by an obstructed state of the alimentary organs. . . . It is a common feature in insanity or semi-insanity left to itself, I think. I also exerted my brain to the extent of abuse, I know, in the way of study. . . . I used to study Latin for a

postime, and often kept engorging my brains over Cicero and Cæsar until the top of my head was very sore. This solitary immersing of an enfeebled mind in study, with obliviousness to myself and all surroundings, was, no doubt, a help toward the grand consummation that took place in the fullness of things. . . . I suffered a good deal from bodily ailments. My liver seemed to be thoroughly out of order and torpid. I had a feeling of hardness and inflammation in my sides regularly, a certain length of time after meals; digestion was bad, appetite irregular—in fact, every sign of a deadlock in the vital functions."

His mother and he removed to another village in 1871, when he was twenty-eight years of age, by which time there was but little question of his insanity, even among his relatives. I let him take the thread of the story again at this epoch:

His mother and he removed to another village in 1871, when he was twenty-eight years of age, by which time there was but little question of his insanity, even among his relatives. I let him take the thread of the story again at this epoch:

"When my mother was making preparations for moving she asked me to help in packing up some chairs. I made an effort to apply myself to the task, but suddenly found myself overcome by my feelings, and before I knew what I was about I had shivered one of the chairs to fragments. A most unpromising omen! The fact is that I was, and had been for some time, in a state which my physician, knowing the facts, would have pronounced to be unmistakable insanity. But I had different ideas about what constituted insanity, and often thought to myself that if I did get put into an asylum, as had been threatened, they would not keep me, because they would see that I was perfectly rational. I have learned more about the subject since."

"Things of the kind I have told of had happened to me before, at uncertain intervals, during several years, an obstructed state of the bowels bringing on a turn. I would get into such a condition of exaggerated discomfort as to lose for a moment, or sometimes quite a spell, my control over my actions, and act very strangely. Sometimes I dashed down an article I happened to have in my hands, or demolished the first thing that came to hand; sometimes I gave vent to my feelings by grating my teeth, 'clawing' my face, and going through strange grimaces and agonizing contortions. My face seemed to me to be paralyzed when I had such turns, as if lifeless. The worst thing I ever did was when I flew at my mother in a sudden access of frenzy one day, when she had wrought upon my feelings by talking to me irritably, and bit out a mouthful of her hair. . . . When I was committed to the asylum, at a later day, it was reported as one of my symptoms that I had delusions about my mother being my enemy, etc., but nothing could be further from the truth. I often grieved in secret over my inability to be a stay and protection to her, benefit as she was of all other support, but all in vain."

"In my new home I was in one of a row of houses, with strangers

living near on both sides, and the sense of the presence of the evil which I had shrunk from so long weighed down upon me with crushing weight. After a while my spell of hypochondriacal despondency passed off, and I settled down into the way of living which I adhered to as long as I remained there. As to getting acquainted with my neighbors, or having any intercourse or dealings with them, that was altogether out of the question.

I now had more of the feeling of constraint, from the knowledge that I was moving under the eyes of people who were strangers to me, than the strangest of the strange could be to a person of the ordinary stamp. Sometimes I heard remarks which did not affect my feelings flatteringly, but that was not common.

"Along in June I had a worse spell than common of the kind of nervous stagnation or will-impotence of which I have spoken, and perpetrated some quite irregular acts before my fetters became slackened. In my despair I tore the collar from my shirt, tore the slippers I was wearing, slashed my fist into a tempting dish which my mother was offering me to eat, and other things of the kind. The house we occupied was owned by a maiden lady who lived with her sister in part of the house.

In the evening, after the other sister returned, who had been absent during the day, I overheard a few words which showed plainly enough that the events of the day were being discussed in no very gratified humor. It was evident that my acts were severely reprobated."

The next day the justice of the peace called upon him and admonished him to restrain himself, hinting of the asylum. Of this our author says:

"The dragon's tooth of reprimand that had been left in my mind grew into a monster, in whose presence I found it impossible to live, and I had a fresh access of despair. It was a hot June morning. I remember seeing a razor and flourishing it, and saying, 'Shave me that razor and I will slaughter him,' or words to that effect, meaning, of course, the justice of the peace."

Both homicidal and suicidal inclinations had long been haunting the secret corners of his mind, for three years before he tells of buying a pistol for the express purpose of making war with himself or some one else. On this day, after meeting the officer, he determined upon suicide. He walked out to two different country stores and bought ammunition. On his way back he passed some men in a field. They all looked at him, and one of them "laughed loud and mockingly, and then cried out, in a sort of squealing way, the intention of which could not be mistaken." Then he played a game of croquet with a young man at his uncle's, and overheard the young man make a covert and derisive remark. He continues:

"I passed the next day in brooding, silent melancholy. It was a rainy day and in accord with my feelings. . . . That night I wrote a little statement to be left behind. . . . It can not be said that I plunged thoughtlessly into the gulf of self-murder. I had from the first gaped the responsibility I was taking on myself, as fully as



my mind was capable of doing it. I felt the whole weight of the condemnation that rested upon me for committing such a deed. . . . I passed some part of the hours of the night in sleep. In the morning my mother came to the door to see how I was, and I grasped her hand with a gesture of agonized despair. She took it as an indication that I was going to have one of my wild spells again, and, as she told me afterward, began to anticipate some work of demolition after I should come down-stairs. After she had gone down, I went and took the pistol from the stand-drawer, put on a fresh cap, got into bed again and propped up my head on the pillows, placed the muzzle of the pistol against the center of my forehead, and fired."

He lost considerable blood from the scalp-wound, but the bullet had glanced off; and, although he now tried to starve himself, he was up and about in a few days as usual, attending to his garden with bandaged forehead. He continues:

"There were some steps taken toward getting me into an asylum after my abortive attempt at suicide, but as there were difficulties about it, and I appeared perfectly sensible and rational, my relatives concluded to let it rest.

"From the time of my shooting until the next spring there was not much that deserves mention. How were my thoughts about suicide? It must be said that I had not totally renounced that idea. . . . I used very often to scan the beams in the wood-house and the coils of clothes-line in the garret. . . . The old difficulty of giving way under the slightest or displeasing demonstrations from others remained as bad as ever. I remember once I was so wrought upon by some trifling thing said or done by one of my relations that I kicked out the bottom of a cane-seat chair I was resting my feet on, in a sudden paroxysm of impotent emotion."

About this time he also made a futile attempt to poison himself by drinking a bottle of strong tincture of valerian that he had made himself. That incident he describes, and then proceeds:

"It was my intention, when I began this sketch of my life, to give greatest prominence to that part beginning with my troubles in Clinton Street—that is to say, the period of confirmed lunacy with hallucinations, according to the world's agreed decision; but it appears at present that my project is not to go into fulfillment. I have been greatly delayed in doing as much as I have by lack of strength.

"To make the account which I have given as full an exhibition of my condition at the time my hallucinations, if such, appeared, I will note some further defects in my mental action which I had noticed up to this time. First, two or three things indicating original lack of control over the brain by the will, or non-identification of my will with the action of my brain, and which I must count for predisposition. I have been troubled from my boyhood with a tendency of my brain to see things it ought not to see in what is placed before my eyes. This refractoriness does not extend to all kinds of fantastic visions, but is limited to the singling out of the lineaments of the human face in the outlines of objects seen. The annoyance I have experienced from this

has varied greatly, according to the state of my health. When I used to be sick with the fever and ague, I would lie in bed and gaze at the coarsely drawn window-shades in my bedroom, until I had made out every possible kind of a profile that could be distinguished.

"The other of the two most serious abnormal peculiarities is the supplying of missing articulations to vocal sounds, heard but not understood distinctly, so as to give my mind the impression of certain words, at the same time that I knew I had not understood. Sometimes I have been really cheated this way, and only found it out by inquiring afterward. This might not give conclusive proof of the deception, it is true. Not to violate privacy, I will illustrate supposititiously. If it were proclaimed aloud, far enough from me to allow the inflections but not the articulations to reach my ear with certainty—

### WE SEE WHERE LIES THE DREADFUL SECRET!

my mind might involuntarily and instantaneously reshape it in such a way that I would understand!

### DISCOVER WHERE LIES WHERE EVER SACKED!

"My attention was always quite easily disturbed by noises, particularly talking. In bedhead the sound of voices in conversation at a little distance after I had retired to rest often gave me very serious annoyance, showing excessive irritability of the brain.

"Such was my mental state on the eve of my being overtaken by a more marvellously awful fate than ever fell to the lot of mortal man.

"My original purpose was to follow the incidents having a bearing on my mental fortunes with tolerable minuteness, in an unbroken chain, up to the time of reaching that wonderful state in which I have existed for the last six and one-half years.

"I shall be obliged to confine myself more to generalities.

"I was in such a towering state of morbid sensitiveness that a slightest tinge of impertinence, insolence, or fancied contemptuousness in the manner of those I met, put me on the rack at once.

It began to occur to me after a little that my ears were becoming wonderfully acute for such things. Very often I would hear lively discussions on my character, and disputes about the proper epithets and titles to be applied to me, which I understood perfectly at an astonishing distance off.

I was wrought up to such a pitch that I formed a resolve that if I were given a sufficiently open provocation, I would attempt a bloody revenge, and on one occasion went out with a razor in my pocket.

I had an oppressive feeling of impotence, as if paralyzed, and suddenly did things I had no intention of doing, as in breaking glass. I had a soreness all through my limbs which I compared to molten fire running through my nerves.

"I began to hear responses to and comments on my performances, and it gradually dawned upon me that I had been making myself a conspicuous object of curiosity to the whole neighborhood.

The comments heard grew more numerous and more and more derisive. I had no suspicion at the time of any of the inspiration being drawn directly from my head. I do not say it was so. This is the debatable ground. . . . It was not until about a week later that it became evident



to me that I was hearing my own thoughts given expression to by foreign wills and voices.

"I heard a great deal about 'inducting,' 'conducting,' 'sphere of influence,' sometimes even 'poles,' positive and negative, and my brain was constantly compared to a magnet. . . . I could find no better explanation myself for a long time than the theory of a fluid, similar to or the same as electricity, uniting brains.

"One was the story of an English physician who had become acquainted with my magnetic properties, and who was on the spot at the beginning, directing the experiment. He was stated to have been the first to form a perfect communication with the inducted brain, and he had drawn off my entire memory back to childhood, and had delivered it verbally in the presence of reporters from the city, who had taken it down. It was stated that the record was preserved in a number of thick volumes. These he had taken with him when he sailed for England during the most prosperous part of the experiment. It was further asserted that he continued in communication with my thoughts, and that wherever he went every one to whom he told the story of the new marvel was also set in connection with the magnetic current flowing from my head, and began to participate in my thoughts. . . . One word more of the English doctor. He is said to have declared that if he had assisted at my birth he would not have suffered me to remain alive, as the monstrous character of my organization could have been seen at a glance. After the whole earth had become pervaded with the magnetism from my head, it would be felt as long as I lived, and the instant of my death would be thus signalled all over the globe, and would be noted and used by all nations as a new era from which to reckon time.

"I would think of the Bible, go and open it at haphazard, and just where my eye fell there was a passage that showed me myself. Once when I had been fretting about my ill success in getting my mother to accord with my views about my neighbors' doings, I hit upon this:

"And it shall come to pass that when my shall yet prophesy, then his father and his mother that begat him shall say unto him, Thou shalt not live, for thou speakest lies in the name of the Lord; and his father and his mother that begat him shall thrust him through when he prophesieth', etc.—Zachariah, xiii.

"But the most perfect identity of all is to be found scattered through the Psalms" [of which he quotes several pages, and then continues]: "I do not intend to appropriate the spirit of these passages, or to make their language my own, but quote them thus collectively as an evidence of fact. I am myself but an inquirer. Do they express the experience of any certain person or persons? Or are they prophetic?

Can it be that the same thing that has happened to me has befallen another in ages long past, and that these are the traces of it?

"I have also found a most remarkably close application of many of the precepts and reflections of Thomas à Kempis in his 'Imitation of Christ.' He seems to keep the same character exhibited in the Psalms in view, only speaking as a monitor, instead of in his person. I presume I find myself mirrored in both these places, because I am an extreme case."

Gradually his delusions, burgeoning one from another, became so

systematized that in the last year of his stay at this asylum he could write in his book :

"The signs are too many and too evident to permit me to doubt that my destiny is bound up with the religion of the world. I steadfastly believe that the words in Jeremiah, 'Take forth the precious from the vile,' are addressed to me; and I can not be recreant to the holiest of duties. I will not waste time in useless discussion, but start with the assumption that it is God's will that I should give the world my opinions.

"If it comes to be generally believed that my sign is a fulfilment of Hebrew prophecy, I would recommend a transfer [of the Sabbath] to the day of the commandment. The very fact of a day one step removed being fixed on by both Christians and Mohammedans looks like an intimation that another step remained to be taken.

"Was it not the confidence of Jesus in the book spoken of above that made him say he knew the Father, when contending with believers in personified derangement?"

Quite a large part of the volume is devoted to expounding the Scriptures, in accordance with his delusion that he is a prophet come to reveal a new religion.

For instance, of Babel he says :

"I find an application for the tower of Babel in my own insane history. I expect a confusion of the speech of the old sects to ensue likewise."

Of Abraham he remarks :

"Abraham is accounted the father of all who believe in the Eternal. I believe I am chosen as his sign for the abolition of all dishonouring beliefs, as Abraham was set up against all idolaters and pagans. . . . I have to note, in connection with the offering of Isaac by Abraham, that I find the date given as 1872 before Christ, coinciding with the year after Christ in which my ear-trouble commenced."

Of Ean :

"We may take Ean for polytheistic religion, recognizing and deifying every force and passion that has dominion over the soul and destiny of man. . . . When it gave up its birthright for belief in a single judge, it pledged itself to go on and submit to be judged by the new master. I believe that the day of judgment has come."

Of the miracle of the rods :

"The rods changed into serpents signify arguments becoming living convictions in the mind of Pharaoh. The evangelists' rods live as serpents in the minds of Christian believers, but I confidently expect that my rod will become a serpent that will swallow them all without trouble.

"Israel is held responsible for the destruction of the heathen and their idols. I conceive that I am the Lord's instrument for the com-



pletion of this work, and that I have been shown these signs in the law that my hands might be strengthened.

"I can not shut my eyes to the fact that I have been made the world's sin-offering."

#### Of the prophets:

"The prophets I will take in a lump, with the assurance that no one can fail to see their connection with my destiny. There is a prophecy in Ezekiel, xxxiii, 33, which is very closely paralleled in my experience. . . . Jonah gives me a parable."

His discussions of theological questions are interesting, perfectly coherent and logical, although often fanciful. He pays tribute to the beautiful moral laws and righteousness of Christ, but is disposed to criticize His conduct as being inconsistent in one who claimed to partake of the omnipotence and omniscience of the Eternal. Of resurrection he says:

"If I conceive of a new body having the memory which I have of this body's life,—and I can find no other idea of the continuance of a soul's life except in the perpetuation or renewal of the memory,—would that in the new body be a *true* memory? Would it not be a hallucination? Would not that be an insane creation?"

In speaking of the years of his greatest mental aberration, he says:

"Here I come to more debatable territory, on which I and the rest of the world have until this present been at variance. I will, in deference to the other side, make use of the word *believe* in stating facts drawn from the region of my memory lying within this shadowy world. I will be permitted to say, therefore, that I believe that after settling down in the before-mentioned place, my brain was, by the gradual progress of events occurring naturally and according to the ordinary laws of human affairs, drawn into relations to the living actors around me, of an altogether unexampled kind—at all events, different from anything plainly recorded in the annals of past ages. I believe that the final result of such relations was the supervening of a state of mental intercommunication through the medium of my sense of hearing.

"But this is a very odd story, and merely a restatement of the perfectly well-known features of my alleged monomania. Let me pass on and give, as well as I am able, my own theory on which I explain these phenomena, which may have more interest. It is a question of personal identification. How does a man use his own brain? He can use it because it recognizes the actions of his members as belonging to the personal unit of which it forms the summit. Now the question is, can not a human brain under certain circumstances become so perverted as to recognize for itself, and without the mediation of its bearer, the acts of other individuals as belonging to its life, as falling within its own memory? And if so, would not these individuals become partakers of the intellectuality of that brain, know its conceptions and ideas, while it thus recognized their motions, and become able to share its walks and ways? Such I believe to have been the result in myself, from the towering height of disintegration reached by my mental organism, by

the gradual process which I have endeavored to faintly shadow forth in the preceding five chapters.

"Let us see whether it does not look probable that a mind in the habit of separating recognized observations from its own responsibility, considering them *objectively*, philosophizing on its own manner of working, driving the impotent and erratically acting part into a corner, as it were, would not be more exposed to such a fate as supposed than one acting unitely, and right or wrong as a unit. It may not be susceptible of argument based on points of organic action, but it looks a plausible thing to me that the insane quality or element in such a brain might be acted on from without, and give itself up to such action, independent of the thinking will of that mind.

"But let us further suppose some little abnormality about the original constitution, a predisposition from a slightly dislocated arrangement of mind-apparatus and sense-apparatus.

"Such, say I once more, I believe to have been the case with myself, and such to be the true nature and essence of the things which have constituted my insanity. . . . I do not deny the fact of insanity, but I firmly believe that it is and has been, since the summer of 1872, an insanity involving the will, ideas, and acts of more than one individual.

"Notwithstanding my full and necessary faith in the reality of things as I have reasoned to prove them, I am still willing to concede that there has been more or less of purely subjective illusion mingled with these dual realities. Under one aspect the whole of this train of mental images and impressions which has whirled through my head has consisted of insane delusion. The effect on the state of my system has no doubt been analogous to that produced by delusions, and the nervous condition which preceded it was such as eventuates in the rise of delusions. Does not the development of delusions often have a compensating effect in invigorating the nervous system in a manner from its trainwheels? Perhaps when this supercomes the brain becomes a chimney for the combustion of the matters which threatened to entirely interrupt the action of the system by clogging. The patient is then known as sensible on most subjects, but a confirmed monomaniac."

Certain peculiarities in his hallucinations possess considerable interest. They almost always referred to the intercommunication of brains. In July, 1878, he wrote out a list of specimen phrases which he had heard while sitting alone at an asylum window. Some of these I reproduce here:

"One thing you know, you know when you get your will in there you get him into a hell of misery."—"He ain't got any will there to feel away."—"Although you are knowing his ideas you connect with her will."—"Instead of connecting with his ideas you keep giving him to her."—"You can't get your will there till he connects his through to his thought."—"We are all the while trying to make him think himself."—"I think we ought to be making efforts to get the idea out on the ball."—"After they get the whole will he is in a hell of torture all the while."—"We keep hollering till we get him into a hell of horrors."—"You see, when there are two wills connected with the head at the same time, he ain't nowhere."



These were the voices of several men and women. In fact, his hallucinations were always polyphonic, and at times would be polyglot. They did not address him directly, but spoke to one another about him. He seldom had hallucinations of hearing except when the ear actually received the sound of distant conversation or inarticulate noises; so that for their production it was usually necessary that there should be transmission of vibrations to the auditory cortical area. As instances of the polyglot character of the voices on occasion, I relate the following:

Once he heard some one call out, "If he ain't a prophet there never was a prophet—*tabulas delit ut vinever*." In tracing this Latin to its source, he found it was a perversion of a phrase in a note to Whiston's "Josephus": "*Ego et tabulas delit ut vinever*" (I myself carried the letter commanding that I be bound, attributed to Bellerophon, which he had once read).

At another time in a street-car, a German sitting next to him cried out, "*Das ist das grösste Mirakel von der ganzen Welt. Jeder Gedanke der ihm in den Kopf gekommen ist hat die ganze Village gehört.*" (That is, the greatest miracle in the world. The whole village has heard every thought that has come into his head.) The grammatical construction of the foreign phrases is open to criticism. The language used by his invisible tormentors was always a peculiar dialect, often abounding in slang, which he considered the most hateful kind of language, and which was such as he never voluntarily used in the composition of his own sentences. The hallucinations were usually boisterously satirical, teasing, quizzical, frequently accompanied by laughter.

**Course and Prognosis.**—The usual course of paranoia has just been outlined. Many cases, however, enter into a state of secondary dementia toward the last.

The prognosis is absolutely unfavorable. I do not know of a single case that has recovered. These patients may live to an advanced age, especially under the fostering care of an asylum. Remissions are occasionally noted.

**Morbid Anatomy.**—The disorder is purely functional. No pathological changes have been found in the brains of paranoiacs. In some instances asymmetrical arrangement of the convolutions has been noted. These belong in the category of stigmata of degeneration.

**Treatment.**—Therapy does little or nothing for the disease once it has become established. Sometimes complete change of environment brings about a remission. Constant physical occupation, hard work out-of-doors, is perhaps the most useful of remedial agents, in that by this means the mind is diverted from the constant contemplation of hallucinations and delusions, and through bodily fatigue is made to receive a considerable amount of repose. Labor acts as a counter-irritant. By it episodic outbreaks of excitement may be aborted or reduced in intensity. Prevention naturally would be of vast importance, were one able to anticipate the coming catastrophe in the prodromal period. Children and youths who exhibit such symptoms as have been described as incident to the hypochondriacal epoch of the evolution of paranoia require a special system of education and training, in which occupation of the muscles and out-of-door life should play the chief rôle.

## CHAPTER XL

## THE NEUROPSYCHOSES.

## HYSTERICAL INSANITY. EPILEPTIC INSANITY.

Under the designation of neuropsychoses are included certain conditions of a hysterical, neuro-thenic, or psychathenic character, as well as mental disorders associated with epilepsy, chorea, Huntington's chorea, and Parkinson's disease. As the mental symptoms and characteristics of these disorders have already been described in previous pages under the headings of the diseases themselves, only the most important are selected here for special consideration.

## HYSTERICAL INSANITY.

The hysterical character gives a certain color to other psychoses when present in a given case. This character, having practically always a hereditary basis, consists in extraordinary suggestibility, instability of mood and activities, great impressionability, enormous egotism, which leads to the desire to make sensations, occasionally a form of negativism (giving rise at times to refusal of food, mastication, etc.), a tendency to simulation and confabulation, and, finally, associated with these psychic symptoms we may have any of the well-known nervous symptoms (anesthesia, hyperesthesia, paralysis, aphasia, pains, clonus, globus, color-blindness, mydriopia, deafness, tremor, and convulsions). The name "psychogenia," or "psychogeny," has been suggested to replace the now meaningless hysteria. Besides coloring at times various other types of mental disease, there may arise on this hysterical foundation episodic attacks of real mental disorder, such as somnambulism, with amnesia, hypomanic excitement, depressive phases (generally with little or no inhibition), and the so-called hysterical twilight conditions (Dissimulzustände).

These last are usually observed before or after grand hysterical attacks, and consist of a hallucinatory delirium, with more or less clouding of consciousness, or of a religious ecstasy, followed by amnesia on recovery. In many cases there is a continuous recurrence of such deliria, with lucid intervals. There is amnesia during the lucid intervals, which disappears in the recurring attacks, so that the same delirious content may be lived over and over again. The alternation is often so marked as to constitute a species of double personality.

The prognosis, naturally, in any case with the hysterical character is very unfavorable. The episodic physical and mental symptoms generally disappear under some sort of suggestive treatment, but they are prone to reappear in some other form.

## EPILEPTIC INSANITY.

Some ten per cent. of all epileptics become insane. Hence the epileptic neurosis in an individual renders him about thirty times more liable to insanity than if he were normal. The psychoses to which the epileptic is subject vary extremely in character. It is my aim to give



here a brief review of these. I shall not consider under this heading forms of mental disorder in which epilepsy or repeated epileptiform convulsions make their appearance in conjunction with the psychic disturbance as the result of a common cause (general paralysis, chronic alcoholism, epileptic idiocy, paralytic idiocy, etc.), but shall limit myself to the class of insanities induced by the epilepsy. It is, first of all, necessary to dwell for a moment upon some of the ordinary features of epilepsy, apart from the familiar phenomenon of muscular convulsion. The epileptic is subject to peculiar symptoms, which are looked upon as the equivalents of convulsive seizures. Among these are sudden brief losses of consciousness. The consciousness may be merely clouded or completely lost. There may be no perceptible concomitant symptoms. On the other hand, the defect of consciousness may be accompanied by some pallor of the face, a fixity of the eyes, or a partial local spasm or movement (strabismus, stammering of a few words, grimaces, lifting the arm, bowing movement of the body, turning of the head, etc.). The disorder of consciousness may be associated with an automatic dream-state, similar to somnambulism, in which complicated impulsive movements take place (automatic continuance of acts begun before the seizure, purposeless running, undressing, etc.). Vertiginous attacks may be the equivalent of convulsions. The aura of an epileptic attack may be in the form of a hallucination. A study of the psychology of epileptics in general gives us a sort of composite picture, to which all of these patients conform more or less closely. The mental attitude of the epileptic is due to a variety of circumstances. In the first place, he has a consciousness of the dreadful nature of his malady. He is in a state of expectant attention as regards the sudden blackness and prostration which are to strike him unawares at any time, in any place, like the lightning from a clear sky. He can never share the social pleasures of his fellows. The schools are not open to such as he. When he becomes old enough to work, he finds that no one wishes to employ him. Every avenue of education, every trade and calling, every road to mental progress, is barred. He is a social outcast, an object of commiseration, a burden to his friends, perhaps a family blemish to be kept concealed. The doctor is called in, and, taking, as a rule, a hopeless view of the case, abandons him to the mercy of the heavens, which further his mental, physical, and moral degradation. In this way the epileptic character is evolved. It consists of a mixture of melancholy, hypochondriasis, emotional irritability, meanness, distrust, misanthropy, mental apathy, and dullness, often combined with morbid religious tendencies and modified by pathological psychic conditions incident to the ravages of the disease itself. These pathological mental states vary from the peculiar psychic equivalents just described to the actual psychoses of divers forms now to be detailed. Epileptic insanity is chiefly a progressive psychic deterioration terminating in dementia. But the progressive degeneration is frequently marked by episodic outbreaks of psychoses under various forms. Among these are transitory hallucinatory and stuporous disorders and chronic epileptic psychoses (under any form, such as mania, melancholia, circular insanity).

**Psychic Degeneration of Epileptics.**—As is well known, severe epileptic attacks are ordinarily followed by a somnolent and stuporous condition lasting from an hour or two to several days. The frequent repetition of such attacks tends to render complete recovery from such mental torpor more and more difficult. As a consequence, we observe a gradual weakening of the intellectual processes. The flow of ideas is retarded and the expression of such ideas along motor lines becomes sluggish; the speech especially has a characteristic slowness; attention is diminished and memory impaired; the concepts and judgments are built up with ever-slackening activity. In this way the epileptic may sink gradually into a deepening simple dementia. In some cases the concepts attended with ethical feelings vanish first, and to so striking an extent that acts of violence, cruelty, brutality, and crime are committed without a single inhibitory effort or a shadow of remorse. These acts often have an impulsive character.

An excessive irritability of temper is a phase of epileptic psychic degeneration. The most trivial incidents may give rise to outbursts of anger and even of overwhelming fury.

The natural hypochondriacal depression of many epileptics is frequently much exaggerated, giving rise to a sort of melancholia colored by mental enfeeblement, and by suspicion, distrust, misanthropy, and moroseness.

Occasionally, in the midst of this progressive deterioration of mind, imperative ideas and acts manifest themselves, and delirious states appear with dreadful hallucinations and delusions of persecution (paranoia-like outbreaks).

These are the marks which distinguish the psychic side of the gradually developed dementia of epileptics. The mental enfeeblement is accompanied, as in terminal dementias generally, by increase in bodily weight, hypertrophy of the subcutaneous fatty tissue, and the gradual effacement of the lines of expression in the features. We thus reach ultimately the condition of

**Epileptic Dementia.**—As intimated, the rate of progress of epileptic dementia is in direct proportion to the number and severity of seizures. There are cases which go on to the terminal stage without some of the peculiar manifestations of progressive epileptic degeneration just described, and others, again, in which these features are prominent. The dementia may be absolute, so that not the simplest concrete memory-picture remains in the vacant mind; the patient needs care in his person and dress, and often has to be guided and assisted in taking nourishment. His sensibilities become so diminished that he is indifferent to stimulation of any sense, and has no perception of the needs of the body as regards the bowels or bladder. He must be cared for like an infant. A persistent sexual instinct often impels him to constant masturbation.

During progress into dementia, we note the intercurrent hallucinatory states already mentioned, and the accessions of anger, with assaults and impulsive actions of various kinds. The motor memories suffer in the end to such degree that all complicated movements are forgotten. This is particularly noteworthy in the use of words, which are separated by considerable pauses. Often even the syllables are thus divided. Finally, the patient loses the power of speech altogether (aside from the actual



aphasic attacks, which are not infrequently observed in connection with severe epileptic seizures).

The course of epileptic dementia is rarely rapid; it usually extends over a period of years. The cause of death is usually accident, status epilepticus, pneumonia, intestinal catarrh, inflammation of the bladder, or some other intercurrent affection. Epileptic dementes exhibit a diminished resistance to diseases in general, and never attain great age.

**Acute Transitory Epileptic Insanity.**—The acute insanity of epileptics develops suddenly before a convulsive seizure, after the attack, or it may occur in the interval between the epileptic convulsions, commonly in the place of a convulsion, as a so-called psychic equivalent. As a rule, both onset and termination are sudden. The duration of the insanity is ordinarily from a few hours to a few days, though the attacks are sometimes shorter and sometimes longer. The symptoms are peculiar and various. The chief characteristic is the clouding of consciousness. The patient's state may be one of complete unconsciousness, though usually consciousness is not entirely lost. It is rather a condition of sub-consciousness or of subliminal consciousness, with stupor. Upon this screen of clouded consciousness there is a play of multiform and bizarre psychopathic outlines—many-hued, terrible, or ecstatic hallucinations; delirium, mutism, incoherence, verbigeration, anxious states, delusions (often of a persecutory nature), or irresistible impulses to assault, destructiveness, homicide, and suicide. Sometimes the fundamental tone of the outbreak is melancholic, more often maniacal, but the most appropriate designation of these acute epileptic psychoses is, perhaps, acute hallucinatory paranoia. There is no essential difference between them, whether the attack be pre-epileptic or post-epileptic, or the equivalent of the paroxysm.

The stupor of epileptic insanity is distinguished from that of other psychoses by marked loss of consciousness, enfeebled attention, analgesia, sudden violence, and confusion.

We sometimes observe in connection with sub-consciousness primary anxious states, resembling precordial dread, with extremely painful sensations of oppression and suffocation in the breast; and much more rarely primordial exaltation, with acceleration of the stream of ideas.

Hallucinations are mostly limited to the visual, auditory, and olfactory senses, chiefly to the first-named. The patient sees wild beasts, specters, flames, the fires of hell, wheels, gigantic threatening objects, falling walls, overwhelming waves of water; or, on the other hand, the golden gates of heaven, the jasper throne, God, and the choir of angels. He hears murmuring voices, clamor and uproar, the thunder of cannon, or the singing of the hosts of heaven, the voice of God, etc. Disagreeable and noxious or pleasant odors may be perceived. A peculiarity of these hallucinations is a certain monotony of character, a general sameness, in great part due to the rather child-like constitution of the mind of epileptics. Their education and mental evolution are so often, from the nature of their malady, hampered and retarded, that they pass through life with the fancy and understanding of a child.

Incoherence of speech and lack of orientation as to surroundings are more marked in epileptic insanity than in any other psychosis.

The motor symptoms vary extremely. Sometimes we note motor inhibition attaining to complete immobility and mutism, lasting for hours, days, or weeks at a time. Such quiescence is often interrupted by sudden explosive acts of violence. Again, in other cases, we observe agitation, restless wandering about, purposeless and impetuous running hither and thither, assaults, destructiveness, and, rarely, complicated acts, like theft and other petty crimes. A condition of religious ecstasy is not uncommon. The patient may feel himself washed to heaven, where he converses with God, Christ, and the disciples.

In some rare instances epileptics are subject to dream-like states of unconsciousness, similar to somnambulism, in which complicated acts are carried out. Like the somnambulist, such patients may seem to be conscious, may comport themselves in speech and conduct in a perfectly natural manner, and in this condition, which may last for hours, days, or even weeks, commit offenses against the law, wander off as tramps, or do some extraordinary thing in following the imperative, childish, silly, or fantastic ideas which control their dream-state.

The disorders of memory incident to transitory epileptic insanity are both interesting and important. There may be, upon recovery, absolute amnesia as regards everything that has taken place. There may be remembrance of much that has occurred immediately after the insanity has passed, with subsequent amnesia. There may be complete amnesia at first, with glimpses of remembrance afterward. There is rarely any persistent recollection of the events of the psychopathic state.

As has been stated, the rule is for these transitory epileptic insanities to exhibit a sudden onset and a sudden termination. The longer the duration, the less abrupt the cessation. The majority of these patients recover, but recurrence is, of course, frequent. Termination in a chronic condition is rare. Occasionally, death takes place from exhaustion, intercurrent maladies, or from a convulsive seizure or series of attacks during the psychosis. Recurrences tend to hasten a psychic degeneration ending in dementia.

The epileptic nature of such insanity as is here described, where the history is not known, is determined by the following characteristics: (1) Sudden onset and abrupt termination; (2) the terrifying or ecstatic nature of the hallucinations and delusions; (3) disturbance of consciousness and stuporous condition; (4) impulsive acts; (5) dream-states; (6) amnesia.

**Chronic Epileptic Insanity.**—Aside from epileptic dementia, the acute epileptic psychosis just described may take a chronic course, or assume a periodic form, with little improvement in the intervals between the exacerbations. There are cases which closely resemble chronic mania in their long course, and others in which melancholia is the predominating feature. The epileptic attacks to which these patients are subject are naturally the distinguishing feature, and a special color is given such cases by the epileptic psychic degeneration. Occasionally a true circular insanity is presented, with its alternating maniacal and melancholic phases.

**Treatment.**—Most cases of pronounced epileptic insanity require commitment to an asylum. Their proclivity to sudden accessions of rage



and fury and to impulsive acts of violence necessitates this course. Where there is simply a moderate amount of psychic degeneration this course is not necessary.

The treatment should be, in the first instance, prophylactic; but, after the development of the psychosis, it consists of a combination of the treatment of ordinary epilepsy with that of the particular type of insanity presented.

Preventive therapy is concerned with the counteraction of the many elements which favor mental deterioration, with the mitigation of the epileptic's early sufferings, with the reconstruction of his environment. It may be called the moral and manual method. The moral part of it is the opportunity for education, regular occupation, and recreation. The manual and hygienic part of it, the acquisition of out-of-door trades or callings—muscular exercise, which in itself serves to reduce the number and intensity of convulsive seizures. I may be pardoned for dwelling somewhat longer on this subject of preventive therapy, and for allowing my pen to go over the same lines which it has traveled so often in past years, because I am convinced that this moral treatment marks the greatest stride in advance made for centuries in the therapeutics of epilepsy. For ages drugs have been exploited as helpful or curative; but, after all, little has been accomplished from the standpoint of *materia medica*. Only of late years has the moral treatment become prominent. As a rule, the epileptic patient was dismissed by his physician with a prescription of uncertain value and possibly a few general directions as to diet. It was not known to the practitioner—or, at least, he did not concern himself about the matter—that the epileptic could gain admission to no hospital of any kind; that he had no associates, occupation, or recreation; that, debarred from the schools, he grew up uneducated, and with a tendency toward retrogression rather than progress; and that, without teaching, reared in illness, suffering from a dreadful malady, neglected in body and mind, he could find shelter at last only in the almshouses and insane asylums, these being the only institutions open to him. Yet, in by far the majority of cases of epilepsy, the attacks rob them for but brief intervals of the capacities for study, work, recreation, and social pastimes, which they possess in common with their more fortunate fellow-men. Hence the adoption of a scheme of colonization of epileptic dependents on the model of the great German colony at Bielefeld, of which the Craig Colony, in the State of New York, is an example. The Craig Colony consists of a tract of nearly nineteen hundred acres of land in the most fertile, productive, and picturesque valley of the State (the Genesee Valley). Upon this are already some sixty to eighty buildings, with accommodations at present for but 840 patients. Over eleven hundred epileptics are now on the list of patients awaiting admission. Here they are to be given an education in the various branches of learning taught in the public schools, to be instructed in every kind of industry, to be treated each and every one for epilepsy, and to be offered a home in a sort of village life, where they will no longer have the feeling of social ostracism, or be debarred from the privileges of intellectual and moral development enjoyed by the rest of mankind.

The out-of-door life in a farming community has already had wonderful results, which may be learned from the annual reports of the colony. It will suffice to say here that the average reduction in frequency of attacks among all the patients has been fully fifty per cent., and that the mental and moral regeneration of the beneficiaries has been truly remarkable. What the effect of such change of environment must be as a prophylactic against psychic degeneration and insanity can not be estimated. We may now briefly touch upon the medicinal and surgical treatment of epilepsy. The old drugs—borax, nitrate of silver, belladonna, and the bromids—have their uses. One is valuable in one case and not in the other; and each patient, where the disease is idiopathic, and no etiological indication exists for the preferment of an especial agent, must be experimented upon with one drug after another for two or three months at a time, until a satisfactory remedy is discovered. Upon the whole, the bromids are most effective as a general antispasmodic for all cases. While the bromids are, perhaps, the most useful remedy we can employ as an antispasmodic in many cases of epilepsy, their exhibition in every case is not advisable. With a considerable number of patients the bromids are entirely ineffectual; with no small number, too, very serious symptoms, such as acute bromism, increase of seizures, and even insanity, supervene upon their use. In many of the cases where actual good is done by the bromids in reducing the frequency and severity of the attacks, the concomitant symptoms are such that it becomes questionable whether the remedy be not, after all, worse than the disease. The writer makes it a practice, therefore, to exhibit the bromids with caution, and never to employ them until the series of less harmful, but often quite as efficacious, remedies for epilepsy have been tried in vain.

There are some new drugs and remedial methods that have come into vogue of late which are worthy of attention. In the first place, there is *simula*, a South American plant of the *lyscop* family, the tincture of which is given in doses of one to two or three drams three times daily. After an experience in many cases for several years, I would say of *simula* that it deserves trial in most cases; that it is perfectly harmless, which can not be said of the bromids, borax, belladonna, and some other drugs; that in a few cases it has been extremely beneficial in my hands, and that in most cases it has no effect at all. *Simula* combined with small doses of bromid acts very well. The so-called opium-bromid treatment of Flesch is of value for many patients, especially in old and obstinate cases where all other agents have proved ineffectual. This treatment consists of the administration of opium for some six weeks, beginning with one-half to one grain three times daily, and increasing gradually until ten to fifteen grains a day are taken, when the use of opium is suddenly stopped, and bromids in large and gradually reduced doses are given (thirty grains four times daily, to begin with). I had used in certain cases of epilepsy for some years codein with considerable success, but this combination of the opiate with bromids is still more satisfactory.

*Adonis vernalis* combined with the bromids, as recently suggested by Bochenew, is an efficient method of treatment, from which, in several



instances, I have had gratifying results. Digitalis, which has properties similar to *Adonis vernalis*, was formerly frequently given in epilepsy, but the new combination seems to be much more efficacious.

There are a few cases of epilepsy in which careful investigation indicates self-intoxication as a factor. In these an excess of ethereal sulphates (indican) in the urine, together with periodical or constant attacks of gaseous diarrhea, are almost positive manifestations of putrefactive or fermentative changes taking place in the alimentary tract. It is remarkable how much benefit may be obtained in such patients by the regulation of the diet (milk and its modifications, lemons, matzoon, soured, rare or raw beef, eggs, green vegetables, and special breadstuffs, like Zewelach, Huntley & Palmer's breakfast biscuits, and Voelt's *biscuits de levain*), by the frequent drinking of hot water and the occasional flushing out of the large intestine by hot water, and by the use of certain intestinal antiseptics, given two hours after eating, with plenty of water (beta-naphthol or salol, gr. x).

The remarkable effect of the thyroid extract upon general nutrition would naturally suggest the advisability of its administration for experimental purposes in some of the nervous diseases which we are accustomed to look upon as due to nutritional disturbances in the nervous system. With this idea in view, I have employed it in a good many cases of epilepsy, in a number with very good effect. Especially noteworthy was mental improvement in several cases of epilepsy with apparently considerable dementia. It is worthy of more extended trial.

Aside from the remedies for the epilepsy just described, we need occasionally to employ certain other drugs for particular conditions, such as status epilepticus, maniacal outbreaks, pronounced melancholic states of terror, etc. In status epilepticus rectal injections of chloral, gr. xx, with an ounce of starch-water, repeated at intervals of two or three hours if needed, give the most satisfaction. In great blood-motor excitement we should use hyoscin, hyoscyamin, or duboisin hypodermatically, in doses of  $\frac{1}{32}$  to  $\frac{1}{16}$  of a grain. In anxious melancholic conditions morphia hypodermatically is, perhaps, the best alluviating agent to exhibit.

The question of trephining must naturally come up in certain cases of epileptic psychosis where trauma to the head is evidently the cause of the epilepsy and psychic degeneration. The following points are to be taken into consideration as a guide in this matter:

1. In the very small number of cases having injury to the head as a cause the epileptic habit is so strong, and the changes in the brain are usually so old and deep-seated, that an operation, as a rule, does not cure, and seldom permanently diminishes the frequency of the attacks.

2. Of miscellaneous traumatic cases, where a surgical procedure seems justifiable and is undertaken, a cure of the epilepsy may be reasonably expected in, perhaps, four out of every hundred cases operated upon.

3. The removal of a cicatrix from the cortex, supposed to be the epileptogenic focus, will naturally be followed by the formation of a new cicatrix in the surgical wound—the creation, therefore, of a new epileptogenic center.

4. The more recent the injury, the greater will be the promise of lasting benefit.

5. In cases of traumatic epilepsy with marked epileptic psychoses (recurrent attacks of rage, fury, violence, destructiveness, etc.) trephining would be justifiable as a possible means of diminishing the severity, danger, and frequency of the unusual attacks, even though the epilepsy itself or the psychic degeneration might not be improved.

## CHAPTER XII.

### IDIOCY.

**Definition.**—In attempting to make a good definition and prepare a classification of idiocy, we meet with much the same difficulties as exist in connection with the allied subject of insanity. The innumerable definitions and classifications of insanity by different authorities are familiar to all students of morbid psychology. Each author feels called upon to be original in this particular, or at least to modify and improve upon the dicta of previous writers. This confusion is quite paralleled in the matter of idiocy; and it is easy to understand why this should be so, for in both conditions we have deviations from the normal mental state of every possible shade and degree, depending upon a most varied pathology. The etiology is complex, and the psychic and somatic symptomatology multiform. There is no wonder, then, that the clinical picture is hard to draw, and the arrangement into clinical types difficult in the extreme. It is impossible to make any comparison between the psychological state of idiots and that of normal children, for the former is not only one in which the mental faculties are diversely undeveloped or impaired as regards their quantity, but there is infinite variation in the quality of the idiot's psychic functions. Likewise it is impossible to contrast the mental organization of the idiot with the intelligence of the lower animals, for the idiot is always abnormal, while the animal is a normal being in the zoological series to which he belongs.

What seems to be desirable in a definition is that there should be expressed in it the condition of mental weakness existing, the facts that the condition may be congenital or acquired, and may be due to a defect or some disease of the brain, and, further, that the condition is one belonging to the developmental period of life. A definition something like the following would seem to me to fairly express these desirable points:

*Idiocy is vacuol foldiness due to disease or defect of the brain, congenital or acquired during its development.*

**Classification.**—As regards classifications, they have been made upon a basis of symptomatology, psychology, etiology, craniology, heratology, and, to a certain extent, of pathology. But it seems to the writer that the time is not yet come for an accurately scientific classification of the forms of idiocy. It is much the best plan at present to adopt an artificial grouping, chiefly clinical, but pathological to the



FIG. 202.—*Thymopexia.*FIG. 211.—*Chromophilic idiocy, with dysplasia.*



Fig. 34.—Cretin and idiotic pair, standing beside normal brother eight years (showing retardation of growth).



Fig. 35.—Idiot and idiotic twin.

Fig. 36.—Idiot with epileptic seizures.





Fig. 226.—Microcephalic idiot, with, redness, ear-nipples, perverted.

Fig. 227.—Hydrocephalic idiot, perverted.



Fig. 228.—Microcephalic idiot.

Fig. 229.—Paralytic idiot.



Fig. 100.—Myosopathic infantile—gutturostomized and a few weeks.

Fig. 101.—Good-natured imbecile—full grown.



Fig. 102.—Two epileptic states.



extent of our latest knowledge. Almost any of the types of the divisions here made use of may be congenital or acquired. The term *idiot* itself is generic, including as it does all degrees of mental impairment in early life. But the variations in degree or intensity of the mental weakness are indicated by the expressions: *idiot*, for the lowest degree of mental disability; *imbecile*, for a higher degree, and *feeble-mindedness*, for the cases of *idiot* in which the psychic faculties have their highest development. There is some confusion in literature as to the exact limitation and application of these degrees. Sollier has made an attempt to distinguish *idiot* and *imbecile*, but his definition of *imbecile* is not tenable, in the opinion of the writer, for he describes a certain small class of *imbeciles* as representative of the whole order. It is to be remembered that in each of these degrees we have many gradations, and the entire series, from absolute *idiot* to a normal state, leads up by progressive stages through various types of *idiot*, several steps of *imbecility*, and numerous shades of feeble-mindedness, until the borderland between the highest degenerate and the normal individual is almost indelible.

The highest group includes a rather well-defined class of feeble-minded: the "backward children," the *retards* *arriérés* of the French, the *tarbieri* of the Italians, and the *Geistig-erschöpfte* of the Germans. The difficulty is not so much in the delimitation of this class, as in the separation of the group of *idiots* and *imbeciles*. It is easy to make the classification on seeing the cases, but to convey to others the differentiation by description is far from being so, because of the many features—physical, motor, and mental—which are concerned in such division. The writer, while employing the term *idiot* often to include all of these degrees, would define the *idiot* proper as an individual able to give little or no care to his person; incapable of intelligent communication, barely able to express his material wants, most awkward and ungainly in his movements, if he moves at all, and presenting marked evidence in his lack of expression, apathetic attitudes, and physical stigmata of degeneration, of the profound stunting of his mental and physical development. On the other hand, the *imbecile* is able to care for his person and dress, attend to his physical wants, comprehend fairly what is said to him, carry out orders more or less intelligently, is often able to speak well (though sometimes speech may be impossible to a very intelligent *imbecile*); if not paralyzed, he has good use of all his muscles; he is not destitute of expression, though the expression may vary from an evil, mischievous, cunning cast of countenance to one of relishing good nature; there are fewer stigmata of degeneration in this class than among *idiots*.

The clinicopathological grouping of the varieties of *idiot* which the writer has found most useful to him in his work at the Randall's Island Hospital for Idiots is as follows:

1. Hydrocephalic *idiot*.
2. Microcephalic *idiot*.
3. Paralytic *idiot*.
4. Epileptic *idiot*.



Fig. 221—Epileptic idiosy.

Fig. 225—Idiosy, with extreme telodysphasia.  
(Length of mouth index, 51.)



Fig. 326—Hemiplegic idiosy.  
(Brain lesion rare.)

Fig. 331—Marsupialid idiosy.



Fig. 332—Idiosy, in a form of dementia from early  
onset in childhood.

Fig. 333—Epileptic idiosy.



5. Traumatic idiocy.
6. Sensorial idiocy.
7. Meningitic idiocy.
8. Myxedematous idiocy, or cretinism.
9. Ataxurotic idiocy.
10. Idiots savants.

It is impossible, in the brief space allotted this subject, to discuss these various forms of idiocy in detail. The reader must be referred to special works and articles on idiocy—to the general works of Down, Shuttleworth, Vucsin, Sollier, etc.—and to the monographs by the writer and others. Hydrocephalic, microcephalic, paralytic, epileptic, and traumatic idiocy are readily recognized by their symptoms or history. Sensorial idiocy is a form due to the congenital or early loss of two such senses as sight and hearing. Properly treated, these patients are capable of normal mental development (Helen Keller and Laura Bridgman). Meningitic idiocy can usually be diagnosed only by autopsy, unless the history or exacerations in the course of the disease demonstrate its origin. Cretinism is a rare form which has been fully described in many brochures in recent years. The ataxurotic form is still rarer. There are only two of these in the Marshall's Island Asylum among many hundreds of idiots.

The term Mongolian idiocy tends continually to reappear in the literature of the subject, especially in English and American publications. I have never been able to convince myself that such a distinction has any justification whatever. I have seen several cases where the features had some resemblance to what might be called the Mongolian face, but all could be classed under one or the other of the above headings, and such physiognomies would seem to be purely fortuitous.

The term *idiots savants* is applied to all such idiots, imbeciles, or feeble-minded as exhibit special aptitudes of one kind or another, always out of proportion to their intellectual development in other directions, and often remarkable as compared with similar accomplishments or faculties in normal individuals.

There are many cases of the kind recorded in literature, and it is not at all uncommon to hear of idiots in our newspapers and museums who are exhibited as musical prodigies, "calculating boys," and the like. Beyond the fact of the existence of such curiosities, and the record of their deeds, there has been little or nothing written in explanation of these phenomena. The psychology of the condition is exceedingly obscure; and even were the psychological processes which underlie special aptitudes understood, there would still remain the mystery of the manifestation of particular talents or faculties in minds otherwise blank or defective.

The aptitudes may be summarized as follows:

Arithmetical faculty, musical faculty, special memories, imitative faculty, modeling faculty, delineative faculty, faculty for painting, aptitude for games (draughts, etc.), aptitude for buffonery. (See article by author on "*Idiot Savants*" in Appleton's "*Popular Science Monthly*,"

December, 1886, in which a history of some remarkable examples is given. See also page 884 of this book.)

**General Etiology.**—There are nearly twice as many male as female idiots. In idiocy due to prolonged or difficult labor, this disproportion



Fig. 202.—Paralytic idiot.

Fig. 203.—Idiot who acquires facility of skill and use of fingers.



Fig. 204.—Two instances: one epileptic and one of unknown origin (both hereditary and parental).

is even larger (three males to one female)—a fact to be explained probably by the larger size of the male infant. The causes of idiocy may be classified as follows:



Degenerative	Hereditary transmission of nervous and mental diseases.		
	Pathological heredity is the form of vitiating diseases or habits (tuberculosis, rheumatism, gout, herpeticism, syphilis, alcoholism, etc.).		
	Sociological factors (extreme youth of parents, extreme age of parents, disproportionate age of parents, consanguinity).		
Adventitious	Gestational	Maternal	Trauma, shock, fright, diseases, maternal impressions.
		Fetal Disorders	Syphilis, heart disease, arteritis, morbid processes in the brain and meninges, twin pregnancy.
	Parturitional		Difficult labor, primiparities, premature birth, asphyxia at birth, instrumental injuries, pressure on the cord.
	Postnatal		Constitutions, cerebral diseases, trauma to the head.
			Febrile diseases, mental shock, starvation, over-pressure at school.

The relations of heredity to idiocy are much the same as those of heredity to the psychoses described in the chapter on General Etiology of Insanity. The statistics available (such as those of Shuttleworth and Beach, Langdon Down, Kerlin, and Piper) seem to show hereditary inheritance in about forty to fifty per cent. of idiots. The stigmata of degeneration, which are so marked in idiocy, are described in an earlier chapter. As regards tuberculosis and scrofula in the parents, the percentages of these authors vary from fifteen to thirty per cent. Alcoholism takes a high place among the causes of progressive hereditary degeneration (nine to sixteen per cent.). The writer has found that hereditary syphilis is a comparatively rarer cause of idiocy than many would suppose, and this is supported by the statistics of the authors named above (one to two per cent.). As regards consanguinity, the statistics show that the proportion of idiotic offspring of consins to the number of idiots is very slightly in excess of the number of consanguineous marriages to marriages in general.

Gestational causes vary, according to the statistics, from eleven to thirty per cent. Parturitional factors (meningeal hemorrhage from prolonged labor, asphyxia at birth, premature birth, pressure on the cord, forceps injuries, etc.) are active in about eighteen per cent. It may be said that forceps injuries are far less dangerous to the child than tedious labor. Among adventitious causes infantile convulsions occupy a prominent position (over 25 per cent.). But we must remember that the convulsions may act as a real cause, by inducing meningeal hemorrhage; or convulsions may be merely an associated symptom of a meningeal hemorrhage or other pathological condition due to some other common factor. Cerebral diseases (meningitis, hydrocephalus, hemorrhage, tubercles, catarrhism, tumor, and abscess) follow infantile convulsions in the statistical table of causes (eight to nine per cent.).

Acute febrile diseases induce idiocy in some six per cent. of cases,

These diseases are scarlet fever, measles, whooping-cough, typhoid fever, small-pox, and diphtheria. How they act is not yet known. Sometimes it is through meningeal hemorrhage induced by the convulsions so common at the onset or during the course of these maladies. Possibly at other times it is through an infectious encephalitis, or microbic embolism or thrombosis. Trauma to the head, mental shock, sunstroke, and "cranning" at school have a small, yet appreciable, share in the production of idiocy (probably two to five per cent. altogether). The author has found, in his own experience, that insanity in children is an occasional cause of idiocy. In the adult such mental enfeeblement after insanity is a secondary dementia, but in the growing child this secondary dementia is preferably termed idiocy.

**General Symptomatology.**—Since idiocy, as well as its varying degrees of imbecility and feeble-mindedness, depends upon some sort of congenital or acquired defect or disease of the brain interfering with its normal evolution, it is clear that the cerebral functions may be all of them more or less involved, and that no particular psychic faculty can be selected as the one whose disorder retards or influences the development of the other faculties. Seguin is, therefore, hardly correct in stating that the condition of the mental faculties in idiots is normal, though diminished, and that merely the will is lacking to give them proper direction. Sollier has given us one of the best and latest studies of the psychology of idiocy.<sup>1</sup> Following Ribot and others, he maintains that the slow development of the cerebral faculties is due to want of attention; that spontaneous attention is caused by affective states brought into action by sensations, and that those young children are the most attentive whose nervous systems are most easily stimulated. Hence the faculty of attention is closely related to the activity of the sensations. The greater the power of attention, the more intelligent does the individual become. In idiocy, owing to the diminution or loss of the power of attention, the perceptions aroused by sensations are more or less indefinite, and the resultant ideas likewise ill-defined. Sensations become more numerous as the organism develops, and the lack of ideas and recognitions becomes more noticeable.

Following somewhat the natural order of such examination, with the excellent work of Sollier<sup>2</sup> as a guide, we first take up the senses, those avenues which lead to psychological development.

**Sight.**—Between seven and eight per cent. of idiots are congenitally blind. It is necessary to determine whether the blindness is due to defect of the visual apparatus or to lack of attention. Blindness does not preclude the possibility of education, for some idiots with defect of this sense may be educated to a moderate degree. When idiots can look, without in reality seeing, the apparent blindness is due to a complete absence of attention. In idiots less affected, a greater variety of objects will attract their attention. In the higher grades of idiocy (imbecility and feeble-mindedness) vision may be as good as in normal

<sup>1</sup> "Psychologie de l'Idiot et de l'imbecile," Paris, 1895.

<sup>2</sup> The author, while differing from Sollier materially in some of his conclusions, is indebted to his work for many of the details of the psychological symptoms of idiocy.



ness. But many present certain visual and ocular defects, such as hypermetropia, defective color-vision, strabismus, nystagmus, congenital cataract, inequality of the pupils, microphthalmos, and the like. In hemiplegic idiocy or imbecility we may find hemianopia, but the determination of the acuity of vision is difficult in this class of individuals. The perception of different colors is often possible in the milder degrees of idiocy. Good binocular vision is uncommon in idiots. The normal child is sensitive to light at birth. The normal child takes pleasure in the sight of objects as early as the eleventh day, the eyes are normally coördinated by the end of the second month, and he begins to distinguish colors correctly at about the age of two years.

**Hearing.**—There is a somewhat analogous condition of the organs of hearing. It is not always easy to determine whether an idiot is deaf from defect in the auditory apparatus or only sensorially deaf. Idiocy of mild degree is not infrequently induced by deprivation of this sense. In the higher grades of idiocy hearing is nearly always normal. Deaf-mutism cannot be considered as common. The normal child hears on the first day, and is pleased with music in the second month.

**Taste.**—This sense is frequently affected. Gluttony is a marked feature in idiocy. It is common for idiots to eat without mastication; many present a previous taste for alcohol. This is especially true of the higher grades. A difficulty in distinguishing the simple tastes (salt, sweet, bitter, and sour) is not infrequently met with in the milder types, as well as in those with great mental impairment. Inversions and perversions of taste are observed. The normal child evinces a sensibility to the taste of salt, sweet, bitter, and sour on the first day of birth.

**Smell.**—In the normal child strong-smelling substances produce mimetic movements on the day of birth. In idiocy the sense may be much impaired, perverted, or absent.

**Tactile Pain and Muscular Sensibility.**—As a rule, sensibility to touch and pain is uniformly diminished in idiocy of all degrees, mostly through lack of attention. There may be complete anesthesia and analgesia, particularly in the lower grades. On the other hand, there are cases in which the sense of touch may be educated to a high degree of delicacy. It is almost impossible to study the muscular sense in idiots, but it is apt to be impaired in proportion to the other senses. The normal child starts at gentle touches on the first day, and manifests muscular sense as early as the eighth week.

**Thermic Sensibility.**—What has been said of touch and pain applies likewise to the temperature sense. But their vasomotor systems are susceptible to the influences of cold and exposure, and their resistance to external influences and diseases is such that many of them die of pulmonary affections. Some become more stupid in cold weather and brighter in warm weather, while an elevation of bodily temperature (fever) is accompanied by evidences of more active cerebration.

**Morbid Movements.**—A small number of idiots exhibit no motility at all, but remain perfectly inert. But the majority are apt to be in constant motion. These movements tend to take on a rhythmic and automatic character. I do not here refer to such morbid movements as epilepsy, athetosis, associated movements, ataxia, and chorea, often

present in paralytic idiocy; not to tremor, found in sclerotic cases; but to a group of automatic or impulsive movements.

These forms of movements are among the most common and striking symptoms immediately noticed in going through an institution for idiots. A very large proportion of the inmates are observed to be in continual motion. As a rule, the most frequent rhythmic movement is an anteroposterior oscillation. The patient, in a sitting attitude, swings his body slowly or rapidly backward and forward. Sometimes the oscillation is from side to side. Occasionally the hands and fingers are rapidly or slowly flexed and extended, and brought up to the face in movements similar to those in athetosis, but differing from them in that they are entirely subject to the will, just as are the oscillations alluded to. Walking to and fro, retreating, dancing, and so on, are more elaborate forms of the same kind of impulsive movement. Similar movements occur in the insane, as is well known, and particularly in conditions of greatly unbalanced mind, such as secondary dementia. They are spontaneous movements, seeming to have no relation to any stimulation of the brain giving rise to a motor expression. Generally the movements cease for a time when any sensory impression, such as the appearance of a stranger in the room or being spoken to, temporarily alters the feeble current of thought or excites the mental blankness which has given rise to the automatic movement. Children and young animals are full of spontaneous movements, undoubtedly due to impressions received at some time during their lives, or, it may be, impressions inherited; and, while these spontaneous movements of children are undoubtedly similar in their nature to the automatic movements of demented and idiots just described, they do not often present the rhythmic character of the latter. It is probable that in the feeble-minded, upon which nerve stimuli seldom make an impression, the simple old motor expressions are retained, repeated, and become habitual or automatic. Automatism of movement is thus a sign of little aptitude or impressibility, so far as fresh mental stimulation is concerned. In the idiot the impulsive rhythmic movements just described may be regarded as the habitual motor expression of the simplest and oldest stimuli; whereas, in the secondary dement, the analogous automatic movements are to be looked upon as reversionists to the spontaneous movements of infancy. The smiles and grimaces of idiots and imbeciles belong to the same category of infantile spontaneous motor expressions.

There is probably a certain amount of pleasure in the movements in many cases, as sometimes they manifest displeasure if prevented from executing them. There is nearly always a difficulty out of proportion to the intellectual development for idiots to perform associated movements with a definite object. They may be able to talk and read, and even write, yet be unable to dress themselves. This is often a fault remediable by education, according to Seguin.

**Right-handedness and Left-handedness.**—Some twelve per cent. of all children, idiot and normal, are left-handed; but while eighty-eight per cent. of normal children are right-handed, only seventy-two per cent. of idiots use their right hand in preference, the remaining sixteen per



cent, being ambilextrous. This peculiarity is said to be present also among criminals.

**Voluntary Movements.**—Many idiots do not learn to walk at all, either because of general debility, inability to learn, or paralysis. In such as do acquire the ability to walk, there is great retardation in its acquisition. This is also true of other uses of the voluntary muscles for the common acts of daily life, such as carrying food to the mouth, and so on. They are either never learned or they are acquired late.

**Organic Sensations.**—The keenness of visceral sensibility is more or less diminished in all idiots. The sensations of hunger and thirst are lessened, though only very rarely absent. The feeling of satiety after a hearty meal is seldom felt by them; so that if left to themselves, they would eat on indefinitely. The necessity of defecation and micturition is not perceived at all by profound idiots. In the lower and middle grades of idiocy it is often difficult to diagnose visceral disease, owing to the bluntness of somatic sensations, and they may die without giving any appreciable symptoms. This masking of disease in idiocy is quite analogous to the masking of disease in various insanities. The feeble-minded and imbeciles not infrequently mislead the physician by exaggeration, concealment, or falsehood.

**Attention.**—The lack of the faculty of attention is one of the chief characteristics of idiocy. Naturally, it varies in degree from complete nullity to a simple diminution of the faculty, but it is always lessened. The fundamental elements of the faculty are deficient. These fundamental elements are: The integrity of sensory impressions delivered to the brain; an emotional state of pleasure, pain, or interest in such sensations; motor expressions in the eyes, face, limbs, or body of the impressions received. There are two forms of attention, according to Ribot and Sollier, one of which is natural or spontaneous, and the other voluntary, established by education. The latter can not exist without the former.

There are two qualities in attention that are of importance—*viz.*, intensity and duration.

Thus, attention is impaired in idiocy by the defective senses, which convey to the brain feeble impressions. The second element, the affective state, is notably lacking in idiots. The motor factor of attention is damaged in idiocy in a great variety of ways (general weakness, paralysis, contracture, epilepsy, chorea, ataxia, automatic and impulsive movements, and the like). The intensity and duration of attention are restricted to the last degree in this class of individuals.

The intelligence and the possibility of education depend directly upon the power of the faculty of both spontaneous and voluntary attention. It is probable that the faculty is localized chiefly in the frontal lobes of the brain. Ferrier considers it proportionate to the development of these lobes, and some very convincing experiments recently published by Bianchi make it quite certain that the frontal lobes are the seat of this faculty. In idiots great lack of attention is coincident with diminutive size of the frontal lobes.

In the low grades of idiocy spontaneous attention is almost null,

and education is impossible. The higher the degree of idiocy, the greater the degree of spontaneous attention presented, which may be so appealed to as to develop it into voluntary attention, with intellectual progress as a consequence. With idiots, as with the lower animals, attention is always connected with the sense most perfectly developed, which, in the former, is that of sight. The attention of idiots is most easily aroused through the eyes. Exercises of the attention may thus be employed in the diagnosis of states of intellectual weakness. We find idiots without attention absolutely ineducable, leading a vegetative existence; others, again, exhibiting both spontaneous and voluntary attention, but in flashes, as it were, of brief duration and faint in nature; and still others more or less capable of prolonged and habitual attention. It is only in the last-named group of individuals that education is to any considerable degree feasible. The education appeals in some to the simplest sentiments only (such as curiosity, selfishness, the desire of reward), in others attention is aroused by appeals to a higher affective order (such as interest, ambition, and emulation), and in still others attention may be aroused and sustained by habit.

Since the power of attention directed to external events is so feebly developed in idiots, it is not surprising that attention to internal happenings, or reflection, should be totally absent in all grades of idiocy.

Kilist regards voluntary attention as habitual and disciplined spontaneous attention, as an adaptation to the conditions of a higher social life, as a sociological phenomenon. When the development of voluntary attention is rudimentary, and the resulting intellectual defect is marked, as in the lower grades of idiocy, there are no serious consequences from the sociological point of view. Sollier calls the idiot *extra-social*, and makes the imbecile quite distinct as *anti-social*, claiming that in the latter there is an undefined amount of voluntary attention, combined with a relative, though perverted, intelligence, which two factors render him often a dangerous member of society. He speaks of the instability of the attention of the imbecile. At one moment it may be faint, at another intense as in normal man. He passes from one subject to another with the greatest ease, a characteristic which may even be observed in his infancy. Serious matters must be continually repeated to him to make him understand. He grasps the first part of a sentence, and forms his ideas from that, without waiting for the sentence to be completed. He frequently interrupts, and there is no time to answer one question before another is put. Sollier further goes on to say that this instability of the attention for external objects or ideas is seen also in the acts of the imbecile, who is incapable of intelligent labor, and accomplishes his tasks, when uniform, by a certain kind of automatism, without due appreciation of the object of his work. When the object is understood, the imbecile believes he can attain it immediately, and, seeing the first step only, is prevented by failure of attention from properly completing the work or doing it at all. He seems to forget that he has begun, and as a consequence, unless watched, may spoil whatever he attempts. Other imbeciles refuse to work, but make themselves very busy and important in watching and supervising the occupations of others.



Sollier calls them *vagabonds*. They wander away not knowing where, marching straight before them, with indifference to the welfare of the friends or relatives they desert; traveling by night and hiding by day; undisciplined, insolent, and mischievous.

This attempt to separate idiots and imbeciles into two distinct classes of extraneous and antisocial is, to my mind, not justifiable. Sollier has here described a certain class of imbeciles only, and the description is very true to nature, but it is only a group which does not merit an especial classification. As regards attention, we should still hold to the terms *idioty*, *imbecility*, and *feeble-mindedness*, as representing degrees of lack of attention, from complete or almost complete absence to mere diminution of the faculty. The adult imbecile, in the middle grade, would have the varying and imperfect attention of a backward child, and his ideas, speech, and conduct would vary with his temperament, with his docility or perversity; in short, with the innate differences of character and individuality, which may be as manifest in imbeciles as in normal children. Imbeciles may and do become vagabonds, uncertain, mischievous, insolent, antisocial; but they may, on the other hand, be good-natured, trusty, docile, industrious. Many of them, too, may show special aptitudes in certain directions. As to education, the difficulties are that in some it is hard to attract the attention, and in others to maintain it.

**Reflection.**—The internal form of attention (reflection of Ribot), in which images and ideas constitute the subject-matter, is quite deficient in the lower grades of *idioty*, but is present in *imbecility* and *feeble-mindedness* in varying degrees. It is never perfectly developed, as in normal man.

**Preoccupation.**—This is absent in profound *idioty* and feeble in the higher grades. A small proportion of imbeciles are capable of preoccupation, but of an indefinite nature, and sometimes taking on the character of a fixed idea. Often their interest is not aroused so much by what benefits and interests mankind in general as by bad actions, criminal or egoistic sentiments that attract their attention and arouse reflection and preoccupation which may result in felony or crime. Many are too selfish to care for the troubles of others, and too stupid to have preoccupations purely intellectual.

**Instincts.**—The instincts in *idioty* are generally defective. The defect may be imperfection of development or an actual derangement or perversion. The instinct of hunger is present in almost all grades of *idioty*, and is so little inhibited that it is often pushed to the extent of gluttony. The instinct of self-preservation is impaired in nearly all, absent in profound *idioty*, ungoverned by proper judgment in the milder forms. In some there is no sense of fear, and self-injury is possible. In others there is a comprehension of danger and an avoidance of it, or possibly an overweening egotism which may lead to a belief in their power to overcome it. Suicide occurs in imbeciles and feeble-minded, sometimes without determinable cause, sometimes as a result of morbid impulses.

Sleep is good among all classes of idiots, while in the lower grades

it may be both profound and excessive. Whether they dream or not depends solely upon the degree of mental development.

The desire and need of voluntary muscular movement varies with the scale of intelligence, being absent in the profounder degrees of idiocy, and approximating the normal the higher the psychic development. The automatic and impulsive movements in some may represent a fulfilment of the normal need, and the extreme restlessness of others is surely a perversion of the natural desire.

The sexual instinct may be absent, impaired, exaggerated, or perverted. It is seldom normal. Idiots of all degrees present many degenerative stigmata as regards the genital organs, more numerous in direct proportion to the mental impairment. Among these anomalies are: cryptorchidism, unilateral or bilateral microorchidia, spurious hermaphroditism, insufficient development of the entire genital apparatus, hypospadias or epispadias; defect, torsion, or great volume of the prepuce; median fissure of the scrotum, imperforate meatus, abnormally large or small labia, excessive development of the clitoris, hypertrophied labia minora, pigmentation of the labia minora, imperforate vulva, atresia of or double vagina, and uterus bicornis. Puberty is often retarded, but occasionally is early; often it is normal. Masturbation is exceedingly common among all classes of idiots of both sexes. In the profound degrees it is automatic; in the higher it is purposive. Onanism *à cleve* and sodomy are frequently discovered among imbeciles and feeble-minded, and sexual perversities of the most shocking nature are not uncommonly manifested in some because of the combination of the strong sexual instinct and absence of moral sensibility.

The instinct of imitation, which is a low form of instinct, and strong in children and many lower animals, is one to which idiots are very susceptible. It is usually a purely instinctive or passive imitation, seldom an intellectual or active imitation. Its intensity depends much, however, upon the scale of intelligence to which the idiot rises. It is very apt to be shown in the form which is concerned with moral contagion; so that the acts and language of the vicious, mischievous, coarse, and vulgar are most willingly imitated. Simulation is very common among the more intelligent classes of idiots.

**Special Aptitudes.**—In the so-called idiots savants we note the development of special aptitudes, occasionally remarkable, more often only noteworthy in contrast to the general mental viciety. These aptitudes are usually in the direction of music, mathematics, the mechanical arts, building, wood-carving, drawing, painting, memory for facts or dates, playing games, and of a low order of wit or drollery. The occasional preeminence of some particular faculty, where all other traits are defective, would almost lead one to believe in a heterotopia of gray matter in some special locality. Music, the most sensual of the arts, seems to appeal especially to this class of individuals. Often the rhythm of it seems to influence the rhythm of their automatic movements, or it soothes their restlessness or stops their cries. Sometimes unteachable idiots are able to retain, recall, and limit a moderately diffi-



ent time, while higher grades may learn to play instruments by ear, though not by note. Next to aptitude for music, that for mental arithmetic is often surprising. There are also occasional instances of the other talents just mentioned, and doubtless the court fools of the past, with their mischievous pranks and quaint remarks, were recruited to a great extent from the imbecile class.

**Play.**—There is a lack in all classes of idiots, and in direct proportion to the degree of mental defect, of that “superfluous activity which is expended in the form of play.” The activity and attention of normal children are mainly developed through play. This avenue of education is, unfortunately to a considerable degree, closed in idiocy. The lower grades, if they manifest a tendency to play at all, do so in a rudimentary and solitary way, and in adolescence still cling to the simple games of infancy. With others, higher in the scale of intelligence, there is still defect of the play instinct, and a proclivity often to prefer games in which noisiness, destructiveness, and other evidence of rather brutal traits are prominent. Sometimes these games are carried on good-naturedly; at others, selfishness, irritability, quarrel-someness, and a more or less ungovernable nature are evinced.

**Civility and politeness** may be taught to many, but naturally with difficulty to the lower grades and to such individuals of the higher as are hard to train in other directions, because of innate vices of temperament and character.

**Destructiveness**, a propensity even in normal children at an early age, is an especial attribute of all classes of idiots. In those of low degree it is automatic and possibly a rudimentary form of superfluous activity (play), but in some individuals of the superior grades there seems, at times, to be a vicious satisfaction in inflicting damage or injury, which may even lead to the manifestation of homicidal proclivities or a tendency to arson (pyromania). Self-mutilation or injury may be a result of the love of destruction in the ponderous degrees of idiocy.

**Sentiments.**—In the lowest forms of idiocy the sentiments and sensations are rudimentary, or may be altogether absent. As a rule, one may discover various degrees of pleasure or pain, affection, pity, fear, social proclivities, love of property, regard for rights and duty, obedience, shame, esthetic feelings, curiosity, and the like.

*Pleasure and pain* are indefinite or absent sensations in idiots, felt to a greater extent by imbeciles, and well marked in the feeble-minded. Joy, sadness, and anger are usually aroused by physical sensations. The self-mutilation of some idiots points to an absence of the pain sense, and idiot women have been known to bear children without experiencing the pains of labor. Idiots often cry out suddenly, burst out laughing, or throw themselves about, which is probably explicable by variations of perception in the somesthetic sense. Moral pain or remorse, usually wanting, is sometimes developed to a slight extent. It is not often that these defectives weep, and if they cry, it is but for some momentary pain or deprivation. They live in the present only, and do not concern themselves about the past or future. In the higher grades it is physical, seldom moral, pain that is taken note of. Pleasure is

as little experienced as pain in the lower degrees, and laughter is as infrequent as weeping. Pleasure is expressed by imbeciles and the feeble-minded by laughter, clapping the hands, or cries, though laughter, even with these, is uncommon. There are, however, certain imbeciles that always have a good-natured smile, and laugh readily and excessively over nothing. Frequently the laughter is a true automatic movement, as infantile spontaneous motor expression.

*Affection* is a sentiment not uncommon in idiocy, though it varies with the degree, being often rudimentary, vague, indefinite, and probably inspired rather by the ministrations to his wants than by the caretaker. It is found that nearly all forms, except the lowest, appreciate kindness and patience, and are repulsed and made unmanageable by brusqueness or cruelty. With certain imbeciles and feeble-minded, where the moral sense is not too much obtunded, true affection for individuals is manifested; but when the moral sense is deficient, affection is elementary or absolutely wanting, so that kindness is either unappreciated or at once forgotten.

There are variations of the same nature in *love* for the family and in *friendship*. Absent in the simplest idiots, it may be shown in greater or less degree in the higher grades. In some it is unstable, changeable, and influenced much by the selfishness of the individual. In others, again, there is a perversion of family love, so that they are hateful and disagreeable to their parents or brethren. It is much the same with friendship. Often wild types of idiocy form in asylums friendships for one another, though they are too often apt to be associations of a sexual nature or for the purpose of combining together for mischievous purposes. A true solidarity of interests or social proclivity is seldom observed. Maltreatment of animals by idiots is usually due to ignorance, but there are moral imbeciles who perpetrate cruelties on animals as well as human beings from pure perversity and love of inflicting pain. The passion of *hate*, when it exists, which is extremely rare, is founded altogether upon a physiological basis. *Jealousy* is sometimes, though infrequently, observed.

*Pity* is quite unknown in all degrees of idiocy. Some are amused or curious and some alarmed at the sufferings of others.

*Fear* is a common sentiment in all types of cases, more common than in normal persons, because of the want of understanding. Often the simplest occurrences inspire fear. On the other hand, when much excited, there are types that exhibit no fear at all.

*Courage* is wanting in all classes of idiocy. *Anger* is apt to manifest itself in all degrees and in every age. It is apt to be both causeless and paroxysmal, and to lead to the infliction of injuries upon the individual himself, upon inanimate things, or upon persons in the vicinity. The ungovernable rage is usually increased by efforts to restrain the patient.

*Acquisitiveness* is shown in imbeciles and the feeble-minded by a propensity for the collection of all sorts of useless objects and trifles, much the same as in cases of chronic mania. There is often a marked tendency to steal, sometimes deliberately, and at other times without



motive, merely to gratify the desire of possession. The lower orders appropriate everything coming in their way, having no regard for the property of others. Many can be taught acquisition as a reward for labor, and, on the other hand, there are some who can be made to work only through fear, having, as they do, an innate antipathy to occupation of any kind.

With respect to *rights and duty*, the perceptions of the idiot vary with the degree of mental and moral defect. In some even inferior idiots these perceptions may be present, while with some the rights of others are never respected, though to their own they may cling tenaciously, and the feeling of duty may never be instilled into them, because of more or less moral perversion.

*Obedience and respect for authority* vary, too, with the amount of intelligence and the degree of moral impairment. Quite simple idiots may quickly respond to the use of command. On the other hand, some of the most intelligent may perversely resist all attempts at discipline. Compensation and punishment affect them variously. Reward in objective shape or in the form of praise is seldom appreciated by inferior grades, and often unduly by the higher. Punishment, objective or in the form of blame, is useless for the simpler degrees of idiocy, where acts are unintentional, and in some of the more intelligent excites antipathy, an unreasonable sense of injustice, and often causes them to harbor a vengeful feeling.

A true *religious sentiment* is quite unknown in any form of idiocy. This is true also of the feeling of *shame*. The only *esthetic sentiment* found in these defectives is the *love of music or rhythm*, which is quite general among all classes, though not perhaps so interiorly as it has sometimes been stated to be. Occasionally we meet with cases having unusual musical aptitude. It is rather a rhythmic noise which appeals to most of them, such as beating of a drum, hammering, the grinding of an organ (even if out of tune and dissonant). They have no sense of beauty, but things bizarre, grotesque, glittering, and colossal attract their attention. *Curiosity and astonishment* are aroused more readily through the sense of sight than that of hearing, and are often more easily roused in some of the lower grades than in the higher types of idiocy.

All classes evince a marked *credulity*, and often it is difficult or impossible to eradicate an idea once established. Fairy stories are especially pleasing to many of them, just as they are to children.

*Veracity* is a virtue which is uncommon among idiots. Many imbeciles are particularly apt to be untruthful and deceitful with regard to their faults, doings, physical condition, things found in their possession, and the like. Naturally, the simple idiot, owing to his feebleness of invention, if given to lying, limits his utterance to the simplest matters, such as denials of accusations brought against him, etc.

**Physiognomy and Expression and Character.**—Idiots all show deficiency in their general appearance. There is always something ungainly, unsouth, ugly in their figures, faces, attitudes, or movements. Very common among them are misshapen or asymmetrical heads, dwarfishness, lack of proportion of the limbs, stooping and

slovenly postures, deformities of the hands or feet, and awkward and wobbling gait. The expression of the face varies from complete apathy and absence of intelligence to a considerable play of features of a low order, such as constant laughing, making faces, lying, or scowling. Besides the absence of those facial traits which are made on the face by the mind, the ugliness is generally added to by asymmetry, disproportion or deformity of the features. The eyes may be too close together or too far apart, or deformed by disease of the iris, cornea, or lids, or by squint. The nose deviates or is malformed, the ears are unsightly and unequal, the mouth half open, the teeth diseased and neglected; the chin deviated, prominent, or retreating; the forehead low and bulging or inclined. Microcephalus, hydrocephalus, and cretinism give their own ugly individuality too well known to need description here. Where a head is shapely and a face has any vestige of pleasing lines, it is generally fair to infer that the mental state is due to deprivation of one or more senses, or to the insanity of childhood.

As to character, this, too, varies with the amount of mental defect, and is difficult to analyze. In profound idiots there are often sudden accessions of excitement without apparent cause. In higher types the basis of character is inconstancy, weakness of will, and blunting of the sensibilities, their humor depending largely upon their environment, showing an appreciation of kindness and resentment of ill-treatment. Some are clever and good-natured and funny, often making sharp remarks or doing amusing things, and at one time such cases were in great demand as court or family fools. History shows there were two kinds of fools made use of by royal and noble families—the true or natural fools (idiots or imbeciles), who were the first to create the profession, and their crafty imitators, the artificial fools, who made of it a profitable calling.

I should differ entirely from Sedgwick in his somewhat extraordinary distinction of imbeciles from idiots. He really selects one type of imbecile, while we know that there are many, and erects this single type into a great class which he everywhere distinguishes in his book as the imbecile. To him the imbecile is egotistical, boastful, vicious, careless, dangerous, a glutton, a vagabond, a mischief-maker, a sexual pervert, unstable, lazy, abusive, obscene, forgetful of kindness, vengeful, shameless, and altogether antisocial.

**Language.**—The primitive physical basis of language in the normal human infant is the auditory tract and the vocal-hearing center. It is essentially receptive. Then develops the word-comprehending center. After this the motor speech center is developed and associated with the primitive physical basis, thus establishing the emissive faculty. This rudimentary linguistic apparatus is variously defective in idiots. A defect in the emissive power is not so serious, as regards intelligence, as one in the receptive; for idiots of considerable intelligence may not be able to talk at all, while others very inferior may speak with readiness. Any part of this original physical basis of language may be affected, and the result to the defective individual will depend much upon what function is lost. The auditory apparatus may be imperfect.



The word-hearing center may not act. The word-comprehending center may be undeveloped. In such instances the intellect will suffer severely. Unlike the normal child, which comprehends many things said to it as early as nine months of age, in cases of this kind comprehension will develop very late, or perhaps never; yet occasionally with the development of the emissive power (without the word-comprehending center) words may be heard, learned, and repeated, constituting an *echolalia*—speech without idea. Supposing the emissive apparatus alone to suffer, we have hearing and comprehension and the development of the mind, yet without the power of speech.

Like an animal, the idiot may be intelligent, but speechless. The development of language and intelligence is not parallel. Sollier distinguishes two kinds of autism in idiots—a motor and a sensory aphasia. In the first he can not talk, though he understands; in the second, nothing which is said is understood. Language is very late in development in idiots. The crying of the normal infant is not often observed, but meaningless and monotonous cries take its place. The laryngeal sounds are earliest and best enounced, the lingual and labial latest and least distinctly. Wildermuth classifies the dysarthrias and halopthias of idiots into two groups:

1. Where the disturbance of speech is the direct expression of the intellectual density. They lack ideas, and consequently have not the words for the expression of them. In the lowest degree, the idiot is a vegetative automaton; in a less profound degree, he is like a child of two or three years, with imperfections of grammar and syntax.

2. When the disturbance of speech is a complication of idiocy, and is mechanical rather than intellectual, Wildermuth has rarely found stumbling speech in the idiot, and never stammering. These defects are sometimes found in imbeciles, who, moreover, talk a great deal and without definite object; who have *conmutosia*, and who are subject to transitory attacks of excessive and unaimed loquacity.

Considerable loquacity is occasionally observed in cases of acquired idiocy.

Next to hearing, the visual tract and the word-writing and comprehension centers form a great receptive system for language and ideas. *Reading* will be impossible to such idiots as have defect of the visual apparatus or these centers, and the degree of acquisition of this power will depend upon the degree of defect. There are idiots who learn merely the letters, others who acquire unmovables, and still others who can be taught to read laboriously. Sometimes such reading is purely automatic, without actual comprehension. The higher the grade of idiocy, imbecility, or feeble-mindedness, the greater the development of this faculty, though few of either class ever attain to perfectly correct reading.

The writing center and its association tracts are the latest portions of the linguistic cerebral basis to be established in normal cases, and in the idiot are apt to be the least well-constituted. In addition to its intellectual side, there is a complicated muscular coordination required in writing which also renders it more difficult for defectives of this kind.

They may be taught to reproduce letters, but the characters are meaningless to them. A few write quite legibly, though seldom or never well. As Sollier says, their writing is in reality drawing, and they like to copy printed letters, curved lines, and so on. There is a certain tendency to write with the left hand and to write from right to left.

In *drawing*, such as learn at all copy slowly and uncerainly, without perspective, and never draw without a copy or model; or they do the work impatiently, and, if given free rein, indulge in curious and fantastic scrolls, such as are figured in the works of Sollier, Bourneville, and others.

**Intelligence.**—Since intelligence depends upon the acquisition, conservation, association, and production of ideas, and these upon the condition of the sensory organs and centres and language centers, it is mainly in intelligence that the idiot deviates from normal man. The deviation varies much in degree, from almost total absence to a condition nearly approaching the normal. The idiot has fewer ideas than the imbecile, and the imbecile fewer than the feeble-minded. All classes require ideas primarily in the same way as the normal infant—through the senses; but while the normal child later on requires ideas chiefly by means of language and imitation, the defective continues to make use mainly of the senses for this purpose, owing to the faulty development of the language centers. Preyer shows that questions and answers are understood before the normal child can speak (nine months), while idiots, many years of age, may have an intelligent idea of the use of things, yet not know their names when heard, and be unable to speak them.

As regards concrete ideas, such as the different qualities of matter, it is noticeable that the idiot appreciates colors (particularly red), recognizes surfaces, avoids obstacles, and notices the difference between round and square, while distances and space are not comprehended. As Sollier correctly says, imitation, which is a source of ideas for infants, does not develop the intelligence of the idiot; for to him it does not furnish an idea, but creates a mechanism. In the superior grades of idiocy imitation creates an idea which is assimilated by the intelligence; but as the intelligence can not retain it, the result is the same as though it had not been assimilated. Still, it is not just to infer, from lack of intellectual expression, that there is complete intellectual inactivity. That ideas may exist in a brain apparently inactive is shown by the phenomenon of intellectual manifestation induced in idiots by severe pain, disease, etc. In other words, the intellectual receptivity of idiots may be greater than supposed, until some irritation occurs strong enough to show that the preceding stimuli have left their effects on the brain centers. Thus, Griesinger reports the case of an idiot who could only speak a few words until he contracted hydrophobia, when he began to talk of events which had taken place several years before.

As regards the *conservation of ideas*, we must remember, says Sollier, that memory is hereditary, organic, or acquired. Hereditary memory is extremely complex and difficult of explanation, but it apparently occurs in idiots. Organic memory, or unconscious memory,—viz., of



associated movements, such as walking,—although sometimes completely absent in idiots, owing to defective nerve centers and lack of attention, is, nevertheless, better developed than either of the two other varieties. For acquired memory, attention is still more a *sine qua non*, and consequently this is the least developed form of memory in idiots. Memory in an idiot develops slowly; at first its existence is shown only by the stimulus of some violent excitement. This indicates that memory exists in so far as the conservation of the image is concerned, but not enough for its reproduction under ordinary circumstances. In a higher degree of the development of memory, the idiot can recall the memory picture by seeing again the original object (memory for food, memory for places). Local memory, which does not act by satisfaction of a natural need, is only found in educable idiots (remembers his own bed, etc.). This memory is fixed by repetition of the sensation, and has not an emotional basis. These varieties of memory are simple, and do not necessitate language. As soon as language exists, a much wider field opens for the memory.

In simple idiots there is no association of ideas. The primitive forms of association, such as fear and the hope of reward, awaken no associated ideas in them, and even in the superior types of idiocy there is no great development of this form of memory.

It is a curious and inexplicable phenomenon that in certain cases of idiocy there may exist particular, specialized memories, such as for musical airs, dates, and numbers, although memory, in its usual and general sense, may be deficient. Indeed, as a rule, the memory is feeble in all classes of idiocy, and even in cases where the memory is fairly well constituted it is ordinarily mechanical, useless to the possessor, automatic.

Naturally, as abstract ideas result from reason, comparison, and judgment, such ideas are absent in the lowest order of idiocy. Profound idiots have no idea of differences of persons or things. Higher idiots may be able to appreciate superficial resemblances and differences, especially of color and form, but the discernment is so faulty that incorrect inferences frequently result.

Superior idiots appreciate resemblances more readily than differences. Simple generalizations may be possible, however, to all classes. In the lower types such generalizations occur only after long instruction, and, once this power is acquired, they may be fairly correct, but in many of the higher they are hasty and often faulty. In educable idiots, even those who can not talk, there is an appreciation of number, and they may be taught to count. Addition is more easily learned than subtraction, and multiplication can only be learned by those with fairly developed memories. Division can rarely be taught them, and neither idiots nor imbeciles can understand problems. The superior orders of idiocy can count automatically, but rarely are able to do so with proper understanding. They can say two and two make four, four and four make eight; but ask them how many are four and three, and they are at sea. To count beyond ten, the number of the fingers, is rarely learned. But there are phenomenal instances where the mathematical

faculty is remarkably developed, as in the cases of the so-called "calculating boys," some of whom, it is true, are normal in other respects, but many of whom are mentally defective, belonging to the category of idiots or imbeciles.

The idea of *time*, past and future, has seldom a place in the brain of the idiot.

Ideas in the idiot are too feeble to be fixed ideas, and while the higher types are sometimes subject to morbid impulses, there is not a true fixed idea, with consciousness and pain. With them such ideas should rather be called *sensuous ideas*.

The association of ideas occurs by resemblance, contrast, and contiguity. In the profound idiots, with few ideas, there may be an association of them in a very simple way—*viz.*, the sight of food is associated with the sensation of satisfied hunger, and so awakens the idea of eating. It is an association of sensations rather than of ideas. The association of ideas should arouse the critical faculty. The judgment and reason in idiots are very faulty. They are founded on an association of few ideas, lack precision and firmness, and find their expressions in ambiguous language. A judgment is not always the result of reasoning. For reasoning, there must be some obstacle to an immediate conclusion. Justice, promptitude, and firmness, which are qualities of judgment depending on the attention, are lacking in the judgments of idiots. The idiots judge very falsely on account of lack of attention and of an association of the simplest ideas. All their sense illusions give rise to false judgments. Firmness is lacking in their judgments, as they have so little interest in what they decide upon.

Many imbeciles and feeble-minded, however, maintain their judgments with tenacity. They often have a very high opinion of their own intellectual faculties. This presumption leads them often to extreme blunders. If one of their judgments is admitted to be just, they become very proud of it, and immediately set to work to form others, which are generally absurd. Doubt which suspends action is rarely seen in any form of idiocy. The first impression capable of forming for them a judgment is followed immediately by the act, like a true reflex. Syllogistic reasoning does not occur either in idiots or imbeciles. Errors of the senses proceed from the perceptive apparatus rather than from the sensory apparatus. Since in idiots and imbeciles sense perceptions are retained in brain centers either undeveloped or diseased, and the sensory pictures are consequently either confused or false, the association of these pictures is consequently faulty. In idiots, as the images are weak, the perceptive reasoning is also weak or wanting. In the imbecile, where the images are more numerous, the association may be falsified by a badly acting perceptive center. In him the association occurs so often by contiguity, and consequently the deduction is very liable to be erroneous, as contiguous ideas are not necessarily related; hence, incongruous observations and unexpected actions.

Sollier emphasizes the difference between idiots and imbeciles, which may be seen in the delirium sometimes occurring in these cases.



Exceptional in the idiot, when it occurs it is always in the impulsive form, unpremeditated and without motive. It is a delirium of acts. In imbeciles there are attacks of maniacal excitement, with inclination to kill, to set on fire, or to break.

With respect to the *production of ideas*, there is little or none in the inferior types of idiocy, and in the higher grades the imagination is inactive, of no utility, and often directed to things that are evil.

**Will, Personality, and Responsibility.**—The older Seguin looked upon defect of will as the basis of idiocy, but the will is rather a diffuse than a local function of the brain. It has no definite seat in the cerebrum, lesion of which would impair or destroy it. As Sollier says, will in its simplest form is manifested by actions accomplished for the satisfaction of natural needs, appetites, and desires. Accordingly, the individual must have a consciousness of these needs. Such a consciousness may be very much blunted in profound idiots, and consequently the will will be almost entirely lacking. Such an idiot is a spinal being, and his movements may be compared to the reflex phenomena seen in decapitated frogs. In higher idiots, the will is manifested by more complex movements, which are, however, capable of becoming secondarily automatic. Voluntary control of the sphincters occurs only in idiots who learn to walk, and not until they have learned. Volitions do not exist in the lowest order of idiots. The most natural desires and the most primitive instincts are absent. The first to appear is desire for food, but it may manifest itself simply by a stretching out of the hand or a cry. In idiots in whom the will is more developed, and also in imbeciles, it finds its expression more easily in actions than in inhibitions.

*Self-interest*, very little developed in the idiot, plays a very important rôle in the psychology of the imbecile, and by catering to it he can often be made to do things which would otherwise be impossible to obtain.

*Intellectual movements*, or acts accomplished under the influence of judgment or reason, are infrequent in the idiot, and not common in the higher grades. Many idiots are incapable of choice. When the power of choice is present, it is often exercised with difficulty. He does not quickly understand that of two things he must take one and leave the other—he wants to take them both. It is the same with ideas. Between two desirable objects, the superior type does not hesitate, but takes without reflection the one he sees first, which he may wish to exchange when he sees the second.

In idiots, whose will and motor volitions are so feeble, suggestion produces little or no results. It is the contrary in many imbeciles, except in those whose voluntary impulsiveness is too great. Ordinarily the higher grades are very susceptible to suggestion, as is seen by the facility with which mischief is done by a band of imbeciles which has been led on by one of their number. If suggestion is possible in imbeciles, it shows that the ideas which they already possess are very unstable, and are easily replaced by new ones. It has a great analogy with the suggestibility of the hysterical.

**Consciousness and Personality.**—As consciousness is but a phenomenon added to psychic processes, and not producing them, and as the personality is the coordination of psychic acts, it is necessary to form by deduction our conclusions as to these two attributes in the class of people we are studying. In absolute idiots it is not probable that any act is accompanied by consciousness. In higher idiots, in whom life is but little more than a succession of disconnected moments, it is not possible to say whether they have consciousness or not; but the personality, if present, must be very rudimentary, since an essential of its existence is a proper appreciation of the continuity of events.

For an individual to have consciousness of a psychic act, it is necessary that the exciting stimulus have a certain duration and intensity. Such factors in the stimuli are generally wanting in idiots; and so it is probable that most of their psychic phenomena occur without consciousness; and if there is consciousness, it must be very feeble. The distinction between the ego and the non-ego is not made by absolute idiots, and is but feebly present in the higher idiots.

In many imbeciles consciousness may be wanting or feeble, but in some it is clearly present, together with a perfect idea of their personality. Further, sometimes in delirium they have ideas of grandeur, showing an exaggerated conception of personality.

**Responsibility.**—All lower types of idiots are unable to manage their own affairs or to enjoy their civil or political rights, but those of a higher degree, who are at liberty, may have these rights.

**Psychological Evolution.**—In every degree of idiocy there comes a time, as Sedgwick well says, when the education stops and further mental progress ceases, and when the only hope is to retain the results which have been gained. This sense of development varies for the different psychic functions, so that one faculty may still improve, while another has already reached its cessation point. The senses continue to develop for the longest time, then the sentiments, and the intelligence the shortest. This is true of all classes, though the periods are longer in the higher grades, where all of the faculties are more equally and proportionally developed. Thus, in inferior types intellectual progress may cease at the age of six or seven, and the sentiments and senses continue their development to eighteen or twenty, while in superior grades the improvement of senses, sentiments, and intellect may cease about the same time—viz., at puberty.

Sometimes the faculties remain stationary, at others they retrograde when the limit of development is reached. Retrogression follows the same law as dementia—namely, progressive enfeeblement of will, intelligence, sentiments, and sensations, in the order named. When retrogression begins in the simpler forms it is very rapid, but in the higher types goes more slowly and more irregularly. Purely intellectual gifts which they have acquired (reading and writing) disappear very rapidly. In the intellectual downfall of the superior types one sees from time to time flashes of intelligence, like reflections from their weakening minds, but such are not observed in the lower forms.

**General Pathological Anatomy.**—There has been accumulated in



literature of late years a great deal of valuable matter relating to the pathology and morbid anatomy of idiocy, so that much new light has been shed upon a somewhat obscure subject. The investigations of Sachs and myself<sup>1</sup> into the causation of the cerebral palsies of children, which are so frequently associated with the various degrees of mental impairment, from feeble-mindedness to profound idiocy, and in which we found meningeal hemorrhage to be so commonly the primary lesion, might well give rise to the belief that in a majority of cases of idiocy without paralysis and in idiocy associated with epilepsy we are confronted with the same initial lesion. The site of the meningeal hemorrhage is the determining factor in the establishment of the symptoms. If the Rolandic area be mainly implicated, either on one or on both sides, we have a hemiplegia or diplegia as the result, and these palsies may be severe or light according to extent of the hemorrhage, and may be associated with idiocy or epilepsy, depending also upon the extent of the lesion and upon the amount of irritation. Again, I have seen a case in which there was left hemianopia, epilepsy, and very slight mental impairment, pointing to a meningeal hemorrhage over the right occipital lobe. Probably, too, some of the cases of arrested development of the speech, with or without enfeebled mind, are due to the same cause. It may be assumed also that meningeal hemorrhage often occurs as the initial lesion in what appears to be idiopathic epilepsy. The symptom or syndrome produced then will depend upon the location and extent of the initial lesion. Asphyxia at birth and convulsions shortly after birth are in themselves significant of meningeal hemorrhage, and in our study of etiology we observe the great frequency of these symptoms in the history of idiocy. At our autopsies, which are nearly always made years after the initial lesion, we find only terminal pathological states, such as atrophy, general sclerosis, and cysts, and, unfortunately, these conditions are not pathognomonic of antecedent hemorrhage, for they also are the terminal states for embolism, thrombosis, cerebral hemorrhage, meningitis, and meningo-encephalitis. What other evidence have we that proves the enormous preponderance of meningeal hemorrhage in the etiology of the terminal pathological conditions? It is in the testimony of the investigators of the causes of still-birth. For instance, Litzmann<sup>2</sup> examined 161 still-born children, finding in them 35 cases of meningeal hemorrhage. Parrot,<sup>3</sup> in 34 autopsies on the new-born, found 5 with blood in the arachnoid cavity and 26 with hemorrhage into the subarachnoid space.

The study of Sarah J. McNair,<sup>4</sup> of New York, in 1885, of 10 similar cases added valuable testimony to that already given, and showed the relation between meningeal hemorrhage and asphyxia and convulsions in the new-born in a manner not to be gained.

<sup>1</sup> "The Cerebral Palsies of Early Life, Based on a Study of One Hundred and Forty Cases," *Jour. Nerv. and Ment. Dis.*, May, 1890. See also paper on same subject by author, Louis Sturt's "Text-book of Diseases of Children," Philadelphia, 1894, and Sachs' "Nervous Diseases of Children," New York, 1895.

<sup>2</sup> "Archiv für Gyn.," Bd. xvi, 1889.

<sup>3</sup> "Clinique des Neuro-musculaires," Paris, 1877.

<sup>4</sup> "Amer. Jour. of Obstetrics."

Allusion is elsewhere made to Herbert R. Spencer's 150 autopsies in still-born children, in which there were 53 instances of hemorrhage from the pia and arachnoid.

Thus, the evidence before us in favor of meningeal hemorrhage as the initial lesion in a large proportion of cases of idiocy is most convincing. Some idea of the character of the terminal states found in idiocy may be derived from the studies of Wilmarth<sup>1</sup> and Bourneville.<sup>2</sup> The former communicates the results of 100 autopsies, which he summarizes as follows:

Sclerosis with atrophy, 12; sclerose tubercule, 6; diffuse sclerotic change, 7; degenerative changes in vessels, ganglionic cells, or nodular substance, not constituting true sclerosis, 15; hydrocephalic, 5; general cerebral atrophy, 2; non-development in various forms, 16; infantile hemorrhage, 1; extensive adhesion of meninges from old



FIG. 335.—Brain of a child with idiocy, showing atrophy of the convolutions over large asymmetrical area—not a true parasymphysis. (See history of case. "Proc. N. Y. Path. Soc." 1894, p. 56.)

meningitis, 3; angiomatous condition of cerebral vessels (with degenerative changes), 1; glioma (with sclerosis), 1; porencephalia (with non-development), 1; of 31 cases where actual disease or imperfect development of the brain proper was not demonstrated, there was hypertrophy of the skull, 6; acute softening (recent), 2; demyelinoccephalic, 2; when the brain was above usual weight, but the convolutions large and very simple in their arrangement, 2.

Our examination of this summary discloses the fact that atrophies and diffuse sclerosis were demonstrated in 21 of the cases and tubercous sclerosis in 6. It is probable that the tubercous form of sclerosis has a pathology different from that of the diffuse form and more resembling the disseminated sclerosis of neuropathologists. Fifteen of Wilmarth's cases are recorded as presenting degenerative changes in vessels, gan-

<sup>1</sup> "Proceedings Ass'n Amer. Inst. Idiots and Feeble-minded," 1899.

<sup>2</sup> "Recherches sur l'épilepsie, l'idiotie," etc., Paris, 1880-1887.



glionic cells, or medullary substance, "not constituting true sclerosis." There was evidently some resemblance to sclerosis, or this author would not have qualified his description thus; and it is more than probable that the condition would have been pronounced one of genuine diffuse sclerosis by experts at the present day. Wilmarth notes 16 cases of non-development in various forms. He writes, in this connection:

"Non-development is found in several forms. A portion of the cortical substance may be thin, and, instead of following the typical arrangement of the fully developed brain, form a number of irregular folds, which may be so small and numerous as to resemble a mass of angle-worms."

This is evidently the condition which we know as *microgyria*, a true pathological process probably due to a vascular lesion (thrombosis or embolism), and not, therefore, a fault of development. Wilmarth's observations were made, many of them, years ago, before neuropathology had attained its present precision, and hence have not the value of later researches, such as those undertaken at Bicêtre and Upsala.

Hammarberg<sup>1</sup> has made one of the most valuable contributions to the study of the pathology of idiocy in literature. His study enters into the details of the examination of the brains of nine cases of idiocy, imbecility, and feeble-mindedness. Several of these were epileptic and paralytic idiots. His pathological investigations were controlled by the microscopic examination of twelve normal brains. The results were briefly as follows: In all of the cases of idiocy a more or less large part of the cortex showed arrest of development at a stage corresponding to either an embryonal period or the period of early infancy. Only a small number of cells reached their higher development or were destroyed during the growth of the cortex. The mental defects were in direct proportion to the defects of the development of the cells, and were greater the earlier the period of arrest of development.

As regards hydrocephalic idiocy, the true pathology of hydrocephalus is unknown. It is generally explained as being due to a chronic intracranial meningitis, a congestion of the ependyma. But in many of these cases nothing abnormal is observed about the ependyma save thickening. It is possible that a careful study of the manner of secretion of the cerebrospinal fluid and of the relations existing between the ependyma and the external serous membrane of the brain may help to elucidate the origin of the disorder; for there is some reason for believing that a sort of current of fluid flows from the ventricles into the exterior serous cavity through the foramen of Magendie, the foramina of Mierzejewsky, and two other foramina which have been described, but are of uncertain existence. The ventricular walls secrete the cerebrospinal fluid and the exterior serous cavity absorbs it, according to this theory. Thus, then, there may be three processes by which primary hydrocephalus may be induced: hypersecretion in the ventricular spaces, occlusion of the foramina mentioned, and disorder of the absorbent apparatus. An interesting study of the subject along this line might be made.

<sup>1</sup> "Studien über Klinik und Pathologie der Idiotie," by C. Hammarberg, Upsala, 1895.

When the fluid begins to increase in the ventricles, these become dilated, as a rule equally, occasionally unequally, from obliteration of the foramen of Monro. The dilatation may be restricted to the lateral ventricles, or may include the third and fourth also. With the distention of the ventricles compression of the brain-substance takes place, giving rise to functional impairment of various kinds and degrees. With increase of pressure, atrophy of the compressed parts occurs. The septum between the ventricles may disappear and the brain-envelope become thin as paper, so that the hydrocephalus is like one enormous



Fig. 322.—Tests of a brain hypertrophic 1864. Atrophy and sclerosis in both cerebral lobes. (See history of case, "Proc. N. Y. Path. Soc." 1864, p. 86.)

cyst filling the cranial cavity. The basal ganglia and brain-stem become flattened. Examination of the cerebral envelope shows atrophy and degeneration of cells and fibers. The distention may go on until the cerebral tissues and the membranes vanish almost entirely. The amount of fluid has been known to reach six, eight, ten, twenty, and even twenty-seven pints. The following is an instance in point (a case from the Randall's Island Hospital for Idiots, the autopsy of which I reported at the New York Pathological Society. See "Proceedings," 1884, p. 94):

A female child, aged eighteen months; hydrocephalus, whether



congenital or acquired unascertained. Circumference of head, 51.5 cm.; anteroposterior diameter, 18 cm.; greatest transverse diameter, 15 cm.; naso-occipital arc, 32 cm.; bioccipital arc, 34 cm.

Blindness and nystagmus; widely gaping fontanel; spastic diplegia; occasional convulsions, and just before death opisthotonos. At the autopsy sixty-four ounces of reddish serum were first removed by tapping the anterior fontanel. The skull and dura were exceedingly thin. The falx cerebri had disappeared. Cutting through the thin dura, nothing was to be seen of any brain proper in the great cavity of the head. The membranes usually covering the cerebrum had disappeared with that organ. At the base of the skull the floor of the ventricles and basal ganglia stood out prominently, and back of these parts, lying on the tentorium, were the only vestiges of a cerebrum—



Fig. 335.—(Left) and (right) in a case of hydrocephalus.

parts of the two occipital lobes. On removing the tentorium, the cerebellum was found to be of about normal size. Microscopical examination showed degeneration and atrophy of the lateral columns of the cord (Fig. 338).

In this case, then, we have to do with distention and atrophy of the encephalon pushed to its greatest extreme.

Case IV, in a series of autopsies by Bourneville, is a good illustration of the nature of the process of compression and atrophy. A girl, a complete idiot, died at the age of about two years. Five hundred grams of fluid were found in the brain-cavity, the brain-envelope having become merely a sac of varying thickness. For instance, in the right hemisphere, over the whole of the temporo-occipital region, the wall of cerebral substance was but a millimeter in thickness, and at one place here, near the fissure of Sylvius, the brain-substance was

absent altogether at a space of four centimeters in diameter, closed merely by a fine meningeal veil. In this case, then, the process of complete atrophy of the brain was arrested by death.

As the ventricular cavities dilate, pushing the brain-envelope with them, the skull-cavity is distended and the cranial bones are separated, made thinner, and expanded in area. The enlargement of the head is directly proportional to the youth of the patient. Cases beginning before or shortly after birth will present greater expansion of the cranial cavity than such as have a later origin. Sometimes some sutures give way and others become synostosed. Where sutures are separated Wormian bones often form, or a membranous connection is established between the cranial bones.

Occasionally, in these cases of primary hydrocephalus, the defects of brain-substance are not due to pressure-atrophy, but there is an associated condition of malformation or defect. Thus, in an autopsy of Bourneville's, on a girl about thirteen years of age, with congenital hydrocephalus, idiosy, and epilepsy, the hemispheres of the cerebellum were usually absent, the cerebellum being represented by the vermis, which was the size of a pigeon's egg. Perhaps such a defect is due to a pressure-atrophy beginning very early in fetal life.

As regards the pathology of secondary hydrocephalus, we possess more definite knowledge. In this the internal hydrocephalus is caused by obstruction of the veins of Galen, or by obliteration of the foramina of Monro, Magendie, or Mierzejewski. Common causes are tumors of the cerebellum, such as sarcomata and tubercles. Meningitis may act in the same way. The amount of hydrocephalus, ventricular dilatation, and expansion of the skull thus induced will depend directly upon the youth of the infant or child. As a rule, secondary hydrocephalus never reaches the extent of the primary form, owing to the rapidly fatal nature of its cause. In these cases we seldom see pressure effects beyond flattening of the convolutions and moderate expansion of the cranial vault.

An exceptional and an extremely interesting case was one upon whom I made an autopsy at Randall's Island, not long ago. It was a case of very marked hydrocephalus in a child of four years, in which a small tumor of the pineal gland, the size of a small hazel-nut, compressed and obliterated the aqueduct of Sylvius. Both of the lateral ventricles were enormously distended, the left more than the right, and contained twenty-four ounces of clear fluid. The third ventricle was also widely dilated. The fourth ventricle was of normal size. Microscopical sections of the quadrigeminal region revealed the obliteration of the aqueduct. The tumor was apparently tubercular, but was not examined, it having been mislaid and lost.

The cases of acute hydrocephalus due to meningitis serosa, and the cases in which a defect of brain-substance is counterbalanced by an equal bulk of cerebrospinal fluid, do not commonly fall under this heading.

In chronic hydrocephalus internus there seems to be a special susceptibility of the membranes to acute disease, so that at autopsy it is not uncommon to find evidence of an acute meningitis, simple, hemorrhagic, suppurative, or tubercular.



The fluid found in hydrocephalic idiots has been frequently analyzed. In a case of Bourneville's the analysis of the hydrocephalic fluid, withdrawn nine hours after death, resulted as follows: Color, pale yellow; aspect, clear after standing; reaction, neutral; odor, like that of blood; consistence, slightly viscous; density, 1.006; organic matter, 1.60; salts, 10; total fixed solids, 11.65; phosphoric acid, 0.22; sodium chlorid, 0.80; albumin, 0.26; leukocytes, very few; red blood-corpuscles, considerable.

In microcephalic idiosy we recognize three distinct classes:

1. Morphological microcephaly, in which there are no pathological changes in the brain, but simply a brain arrested in its development with persistent fetal morphology.

2. Pathological microcephaly, in which the small size of the head is determined by morbid processes in the brain (such as meningeal hemorrhage, thrombosis, porencephalic defects, etc.).

3. Mixed cases of microcephaly, in which pathological processes are superadded to or associated with true morphological microcephaly.

The following table gives a summary of the pathological conditions responsible for most cases of idiosy:

ETIOLOGICAL FACTORS.	PRIMARY LESIONS.	USUAL COMPENSATORY CHANGES AT DEATH.
Hereditary degeneracy.	Developmental defects of portions of the brain, such as corpus callosum, one hemisphere or part of a hemisphere.	Same, with compensatory hydrocephalus internal, external, or both; compensatory thickening of skull.
Hereditary degeneracy.	Microcephalus, with or without defects.	Same. Brain-sclerosis often sclerotic; deficient in subcortical elements. Sometimes compensatory hydrocephalus.
Hereditary degeneracy.	Agenesis corticalis; slight changes in gross appearance of brain; maldevelopment of microcephalic elements.	Same. Sometimes hydrocephalus external.
Vascular disorders of fetal brain.	Partial defects (like porencephalia, microgyria).	Same. Compensatory hydrocephalus and thickening of the skull; atrophy and sclerosis of affected convolutions or lobes.
Disease of mother or foetus to mother. Fetal disorders, such as syphilis, asphyxia at birth, prolonged labor, infantile convulsions, febrile diseases of child, cerebral diseases of child.	Meningeal hemorrhage; thrombosis; embolism; cerebral hemorrhage; meningitis; meningo-encephalitis.	Atrophy; diffuse sclerosis; cysts; meningo-encephalitis.
Uncertain fetal and post-natal causes.	Tumor sometimes; often unknown.	Hydrocephalus.
Antecedent infectious diseases of mother or child (?)	Tuberculous sclerosis.	Tuberculous sclerosis.

In anurotic idiocy but six autopsies have been made, and thus far the changes found may be considered to be simply degeneration of the gray matter of the cortex and of the anterior horns of the cord (Sachs).<sup>1</sup>

**Diagnosis and Prognosis of Idiocy.**—**Diagnosis of Idiocy in General.**—It is seldom difficult to make a diagnosis of idiocy in childhood when the individual has reached such a stage of development that backwardness and deficiency stand out in prominent contrast to the normal average of intelligence in children of the same age. Occasionally, however, we have to deal with some species of insanity in childhood, in which case the matter of diagnosis is important because of the more favorable outlook for insanity. There are not a few patients cared for in institutions for the feeble-minded and idiots in which insanity has been the original factor in the mental impairment, and when the histories of such are obscure, it is almost impossible to distinguish between ordinary idiocy and what may be truly termed a terminal dementia following upon some acute insanity of childhood. In these cases residual symptoms of a psychosis can be our only guide.

The diagnosis of some form of idiocy in infancy is far from easy unless one familiarizes himself thoroughly with the manifold steps of development for the first few years of existence. Early diagnosis is of the utmost importance, not only for the benefit of the unfortunate child itself, but on account of the deep solicitude of the parents for its future. One of the chief aids in differentiation will be found in a study of the physical condition of the infant. The shape and size of the head should be carefully noted and compared with normal shapes and statistics. Unfortunately, there are no elaborate tables of head measurements in infants and children as yet made which can be looked upon as a final establishment of the normal averages, but the following figures are fairly representative of cranial measurements:

Circumference at birth . . . . .	35 cm. in both sexes.
Bregmatic arc . . . . .	22 " " "
Naso-occipital arc . . . . .	22 " " "

At the age of one year these dimensions have increased to—

Circumference . . . . .	44 cm. in both sexes.
Bregmatic arc . . . . .	27 " " "
Naso-occipital arc . . . . .	30 " " "

Malformation and asymmetry of the head should be taken into consideration. The various malformations are treated of in another chapter. The presence of marked anatomical stigmata of degeneration is of significance. Paralysis of a limb or limbs, if of cerebral origin, is of great importance, indicating, as it does, some lesion of the brain, which may retard or restrict mental development and lead to paralytic or epileptic idiocy, or both. Some of the morbid movements, such as nystagmus, ataxia, chorea, or athetosis, may be present, and, as symptoms of dis-

<sup>1</sup> "A Case of Anurotic Family Idiocy with Autopsy," by Frederick Peterson, M. D., *Jour. Nerv. and Ment. Dis.*, July, 1895.



order of the central nervous system, should lead to a careful investigation of the whole mental and physical organization.

While it is frequent to find evidence of idiocy immediately after birth in bodily and especially in cranial and facial characteristics, yet after careful examination as to imperfect action of the sensations and perceptions, we may sometimes recognize idiocy in cases where physical evidence is wanting. The child may not learn easily to take the breast. Its cry is different from that of other children. It cries without motive. Sometimes there is congenital blindness or congenital deafness. In the normal child the sense of smell may be stimulated immediately after birth, and taste is evident on the first day. In the idiot these special senses may be retarded in their development or absent. The movements of the eyes are generally irregular, and strabismus is frequent until the end of the second month in normal children, so that in the diagnosis of idiocy this cannot be relied upon as significant unless the eye-movements are imperfect after the third month. In the normal child the eyes follow a light between the third and fourth weeks; in idiot this ability may be retarded indefinitely. The normal child starts at gentle touches on the day of birth. The new-born idiot may be immobile or feeble in its reactions to cutaneous stimuli. The normal child laughs at tickling in the eighth week, while the idiot or imbecile is not incited to laughter ordinarily at all in the earliest years of life. From these facts it follows that in defectives we must examine the sensory organs themselves, so far as possible, for defects, as well as study their reactions and acquired perceptions of sensations.

Preyer, in his work on "The Mind of the Child," gives a conspectus of the development of the normal faculties during the first forty months of the child's life, and the following brief abstract is made therefrom for purposes of comparison with the mental development of the idiot. This abstract has been modified, however, by comparison with the many studies of child development made since Preyer's time, and particularly in regard to the development of the senses, in the light of the study of 1060 new-born babies at the New York Lying-In Hospital, by Miss Rainey and myself, during the year 1910. (See "Bulletin of the New York Lying-In Hospital," 1911.)

#### NORMAL CHILD.

First month.—Sensitive to light at birth. Pleasure in light of candle and in bright objects on eleventh day. *Hears* at birth. Discriminates sounds last two weeks of month. Starts at gentle touches at birth. Sensibility to taste at birth. Strong-smelling substances produce mimetic movements at birth.

*Pleasure* first days in nursing, in bath, in sight of objects.

*Discomfort* first days from cold, wet, hunger, tight clothing.

*Smiles* on twenty-sixth day.

*Tears* on twenty-third day.

*Facial sounds* in first month.

*Movements* first active as to taste and smell; then as to touch, sight, hearing.

*Inordinate movements of the eyes.* Fixation may occur in second week.

Sleeps two hours at a time, and ordinarily twenty hours in twenty-four (Lying-In Hospital Report).

Reflexes active.

Second Month.—Stimulants occasional until end of month. Recognizes human voices; turns head toward sounds. Pleased with music and with human face. Sleeps three, sometimes five or six, hours. Laughs from tickling at eighth week. Claps with its four fingers at eighth week. First consonants from forty-third to fifty-first days (*ma-a-a*, *ba-ba*, *pa*, *wa*).

Third Month.—Sixty-first day, cry of joy at sight of mother and father; eyelids not completely closed when child looks up. Accommodates at ninth week. Notes sound of watch at ninth week; listens with attention.

Fourth Month.—Eye-movements perfect. Objects seized are moved toward the eyes. Grasps at objects too distant. Joy at seeing self in mirror. Contraposition of thumb in grasping at fourteenth week. Head held up permanently. Sits up with back supported at fourteenth week. Beginning to imitate.

Fifth Month.—Discriminates strangers. Looks inquiringly. Pleasure in crumpling and tearing newspapers, pulling hair, ringing a bell. Sleeps ten to eleven hours without food. Desire shown by stretching out arms. Seizes and carries objects to mouth. Consonants *t* and *k*.

Sixth Month.—Raises self to sitting posture. Laughs, and raises and drops arms when pleasure is great. "Cries" with pleasure. Compares image of father in mirror with original.

Seventh Month.—Astonishment shown by open mouth and eyes. Recognizes nurse after four weeks' absence. Sighs. Imitates movements of head, of pursing lips. Averts head as sign of refusal. Places himself upright on legs.

Eighth Month.—Astonishment at new sounds and sights; at imitations of cries of animals.

Ninth Month.—Stands on feet without support. More interest shown in things in general. Strikes hands together with joy. Shuts eyes and turns head away when something disagreeable is to be endured. Fear of dog. Turns over when laid face downward. Turns head to light when asked where it is. Questions understood before child can speak. Voice more modulated.

Tenth Month.—Sits up without support in bath and carriage. First attempts at walking at forty-first week. Beckoning imitated. Missed parents in absence, also a single needlepin of a set. Can not repeat a syllable heard. Monologue and hints at imitation (*aa*, *pappa*, *baba*, *appa*, *baba*, *bibi*, *pa*, *eee eee*).

Eleventh Month.—Screaming quieted by "sh." Sitting becomes habit for life. Stands without support. Stamps. Syllable correctly repeated. Whispering begins. Consonants *b*, *p*, *t*, *d*, *m*, *n*, *r*, *l*, *g*, *k*, vowel *a* most used, *i* and *e* rare, *u* very rare.

Twelfth Month.—Pushes chair. Can not raise self or walk without help. Obeys command, "Give the hand."

Thirteenth Month.—Crawls. Shakes head in denial. Says *papa* and *mama*. Understands some words spoken.



Fourteenth Month.—Can not walk without support. Raises himself by chair. Imitates coughing and swinging of arms.

Fifteenth Month.—Walks without support. Laughs, smiles, gives a kiss on request. Repeats syllables. Understands ten words.

Sixteenth Month.—Runs alone. Falls rarely.

Seventeenth, Eighteenth, and Nineteenth Months.—Sleeps ten hours at a time. Associates words with objects and movements. Blows horn, strikes with hand or foot, gives leaves to stag, waters flowers, puts stick of wood in stove, washes hands, combs and brushes hair, and other imitative movements.

Twentieth to Twenty-fourth Month.—Marks with pencil on paper, whispers in reading newspaper. Very few expressions of his are recognizable. Executes orders with surprising accuracy. Tries to sing and beat time, and dance to music.

Twenty-fifth to Thirtieth Month.—Distinguishes colors correctly. Sentences of several words. Begins to climb and jump and to ask questions.

Thirtieth to Fortieth Month.—Goes upstairs without help. Sentences correctly applied. Clauses formed. Words distinctly spoken, but influence of dialect appears. Questioning repeated to weariness. Approximates manner of speech to that of family men and women.

By contrasting the mental development of the supposedly abnormal child with these observations upon normal development, it will not be difficult to appreciate impairment of varying degree. The presence of mere backwardness may not infrequently, however, be observed in children that later develop normally, and it is well to bear this fact in mind; but the combination of backwardness in the development of the sensations, perceptions, ideation, and speech with marked physical signs of degeneracy or brain lesion would be naturally of the greatest importance from the diagnostic point of view.

Diagnosis of the Form and Nature of the Idiocy.—While the diagnosis of the presence of idiocy is, as a rule, fairly easy, especially after infancy has reached the stage of childhood, the diagnosis of the type or kind of idiocy presented is often attended with great difficulty. Where the cerebral disorder or defect is accompanied by striking physical peculiarities or malformations, such as hydrocephalus, microcephaly, paralysis, or myxoloma, we are immediately in a position to classify the type. In idiocy associated with epilepsy, too, we can readily approximate the type, though it must always be remembered that there are three distinctive ways in which epilepsy and idiocy are correlated—viz., paralytic idiocy combined with epilepsy, epileptic idiocy from a homologous lesion not implicating the motor centers or tracts, and, finally, dementia in childhood depending upon the epilepsy. The traumatic class of cases is recognized either by the external evidence of injury to the skull or by the history of direct relation of the psychic symptoms to the antecedent trauma. The sensorial type of idiocy is distinguished by existing or forgotten loss of two or more senses, particularly blindness and deafness. The amnesic type presents a characteristic syndrome—viz., flaccid or spastic weakness or paralysis of the

whole musculature, diminished or exaggerated tendon-reflexes, distinctive changes in the fundus leading to optic atrophy, and marasmus. In the majority of cases, then, we are in a position to determine readily the form of idiocy presented by the patient and to formulate an opinion as to the nature of the pathological process or the condition underlying it; but there will still remain a considerable number of cases in which diagnosis can not be made during life, either as to the type of idiocy before us or as to the character of the process. Among such puzzling cases will be those indistinguishable from the psychoses of early life; idiocy following meningeal hemorrhage and meningitis without inducing either paralysis or epilepsy; idiocy due to tuberculous sclerosis, and the like.

**Diagnosis of the Degree of Idiocy.**—It is necessary, for purposes of melioropological treatment, to comprehend the degree of idiocy, not only to determine whether it is simple idiocy, imbecility, or feeble-mindedness, but to ascertain, as far as possible, the different shades of each of these; and it is useful, too, to watch the progress of a case under treatment, and to record from time to time the advance made by the patient and pupil. Accordingly, the writer has drawn up what may be termed a species of mind chart, as given opposite. The physician will be familiar with the ordinary tests for common and special sensibilities. The intensity and duration of attention may be studied, in the same connection, by methods which will readily suggest themselves in relation to objects, colors, sounds, smells, and tastes, which are utilized in such a way as to demonstrate perception, the retention of the perception, and the duration of such retention. The chief difficulty will be in determining and recording the purely intellectual features of the case; but some patience and perseverance will demonstrate the ability and degree of ability of the patient to acquire, conserve, associate, and produce ideas, concrete and abstract; to appreciate resemblances and differences; to count, add, subtract, and divide.

**Prognosis.**—As regards the cure of idiocy, there can not be any difference of opinion. There are few cases—indeed, almost no case—in which improvement to some degree may not be permitted under proper conditions; but cure there is none. The profound idiot may be regenerated to some slight degree; he made less repulsive, less offensive, less destructive. The imbecile can be taught cleanliness, speech, divers occupations. The feeble-minded subject is susceptible of enormous improvement. It is impossible in any case to predict how much advance may be made under the best supervision, but it will be safe to say that the methods now in vogue in the training of the idiot will surprise the relatives or guardians by their efficiency, and there is no case so unpromising and hopeless as to contraindicate an attempt at improvement. Left to itself, even a mild type of idiocy will not only make no progress, but will be certain to degenerate, to lapse into a lower grade. Smithworth,<sup>1</sup> in reviewing the results of twenty years' experience at one of the large English institutions, states that of patients discharged

<sup>1</sup> Take's "Dictionary of Psych. Med.," p. 675.



## MIND CHART.

Name \_\_\_\_\_ Age \_\_\_\_\_ Sex \_\_\_\_\_

Constitution (feeble, fair, robust, or strong) \_\_\_\_\_

Form of limbs \_\_\_\_\_ Degree of limbs \_\_\_\_\_

Pain, deformity, or morbid movements \_\_\_\_\_

Right- or left-handed \_\_\_\_\_ Temperament (cheerful, gloomy, restless,

sluggish, etc.) \_\_\_\_\_

Sense-defects.	Sight.	Hearing.	Taste.	Smell.	Tactile and pain.	Muscular.	Thermal.
Intensity and duration of attention.							
Instincts.	Hunger.	Self-preservation.	Sleep.	Voluntary movements; play.	Sexual.	Imitation.	
Morals and habits.	Tidiness.	Destructiveness.	Humanity.	Voracity.	Politeness.	Obedience.	
Sentiments.	Pleasure and pain.	Affection.	Fear.	Anger.	Aquiescence.	Shame.	Curiosity and astonishment.
Language.	Speech.	Reading.	Writing.	Gesture.	Drawing.		
Intellect.	Ideas.	Memory.	Association of ideas.	Reason.	Judgment.	Will.	Arithmetic.

Special aptitudes \_\_\_\_\_

therefrom after full training, 10 per cent. became self-supporting, another 10 per cent. might have become so had they obtained suitable situations, and about 20 per cent. were reported as useful to their friends at home. This bears out the earlier estimate of Seguin, who said that "more than 40 per cent. have become capable of the ordinary transactions of life under friendly control, of understanding moral and social abstractions, of working like two-thirds of a man; and 25 to 30 per cent. come nearer and nearer the standard of manhood, until some of them will defy the scrutiny of good judges, when compared with ordinary young men and women."

There are certain features in connection with the different types of idiocy which are helpful in forming our opinion as to the probable future of a patient. For instance, it may be taken as an axiom that the greater the defect or injury of the brain, the profounder will be the mental impairment and the more difficult will be the labor of bringing about an amelioration of the condition. The earlier, too, that the brain is hampered in its development, the worse, as a rule, is the prognosis. This holds good for every form of idiocy. Hence the outlook for the congenital types is less promising than that for the acquired, and for idiocy acquired in the first year less than that for idiocy acquired in the second. Some of the prognostic indications of the special forms will be discussed under their respective captions; but, in general, it may be assumed that microcephalic idiocy and congenital hydrocephalic and paralytic idiocy will be benefited least among the types of idiocy discussed, and always in proportion to the intensity of the morbid process. The sensorial, traumatic, and myxoedematous forms are, *et cetera paribus*, among the most promising. The aneuriotic form is generally fatal. Idiots with special aptitudes, or *idiots savants*, tend to early psychic degeneration. Idiots that are extremely restless, as shown by incessant motion of the hands, arms, head, trunk, or by constant walking, are generally among the most intractable, because of the difficulty of fixing their attention.

Although there is scarcely ever to be encountered an idiot in whom improvement of some kind can not be brought about by assiduous cultivation of whatever residual faculties and functions he possesses, it is practically necessary to classify idiots into teachable and unteachable. It is practically so because a majority of these defectives are found among the poor, who can not command all that the world affords in the way of treatment, care, and training. Nor could the commonwealth assume the enormous task of doing the best for all its idiot charges. No community could possibly be repaid for any such undertaking, because the idiots classified by public authorities as unteachable are not susceptible of such development as would satisfy the tax-payers' right to ask the utility of the expenditure. It is only with private families that anxious parental solicitude will and can demand that medicopedagogical care, skill, and patience which can surmount almost insuperable difficulties in the education of profound idiots. Practically, therefore, we find that there is a tendency to separate idiots into the teachable and unteachable; a tendency in our public institutions to exclude un-



promising cases, such as epileptic and paralytic idiots, idiots with malformations, marked cases of hydrocephalus and microcephaly, and, indeed, any patient requiring that particular and assiduous care which it is not in the power of the commonwealth to give.

The prognosis as regards life depends directly upon the degree of injury to or defect of the brain. In general, idiots are short-lived. Diplegic and paraplegic idiots seldom attain the age of twenty years; hemiplegic idiots may live much longer, though it is infrequent for them to attain the age of forty and more years; hydrocephalics perish still earlier. The same is true of profound cases of microcephalic and myxodematous idiocy. The rare form known as amniotic idiocy is almost invariably fatal in infancy.

**General Treatment of Idiocy.**—The treatment of the idiot involves the employment of both physician and teacher. The adjective *medicopedagogic* is made use of to designate this combination of medical and educational features for the care of the defective classes. In the union of the two professions for such purpose the educator occupies relatively the higher and more important position. The institutional services of trained care-takers or nurses are not to be overlooked. That patient will profit most who receives the properly combined aid of the best physician, best teacher, and best nurse. As a rule, this fortunate concurrence of necessary aids is more apt to be found in the public or private institution than in the home; but that it is possible to carry on treatment at home under favorable circumstances, is not to be gainsaid.

The methods of procedure formulated by Itard, expanded by Seguin, and employed now-a-days everywhere in private and public institutions for idiots, with modifications induced by experience and the progress of educational science, are well described in the writings of Bourneville, Shattlenorth, Ireland, Dunn, and others. A brief résumé is given below of the process of

**Education of Idiots.**—The educational treatment should begin as soon as the diagnosis of defective intelligence is made. It need not be pushed vigorously at too early an age; but infancy, when the nervous system is most impressionable, plastic, and pliable, is the time for easy modification and the bringing out of the rudimentary psychic processes which are the foundations for the later conduct, habits, intelligence, and speech. Patients are admitted to the Bicêtre and Salpêtrière at the age of two years and over.

In order to understand the methods of pedagogic treatment of idiocy, let us imagine an infant brought before us afflicted with a profound degree of idiocy—*i. e.*, one showing little or no attention, unable to walk, to use its hands or to speak, and uncleanly in habits. In undertaking a case of this kind the process of education is pursued with the following distinct purposes in view:

1. To develop the attention and sharpen the five senses.
2. To develop coordinated movements and strengthen the muscles.
  - (a) To teach to walk.
  - (b) To teach use of the hands.
3. To inculcate habits of cleanliness in person and dress.

4. To teach the patient the use of language.
5. To arouse the intellect by inculcating ideas of length, weight, surface, solids, form, number.
6. Finally, to carry the education higher, by means of studies in natural history and all sorts of manual and industrial and moral training.

Naturally, some of these purposes are attained at the same time to a considerable degree by some one process employed in education. Thus, when a light bean-bag is thrown at the face of our patient, the attention and sensibility may be so feeble that it is not noticed at first. By frequent repetition attention is developed, sensibility becomes more acute, a reflex movement to ward off the missile is aroused, and gradually, by successive stages, the patient learns to catch the bag, to throw it back, and, finally, to go through a simple drill with it, accompanied by music. This single experiment then improves the attention and several of the senses, and aids in developing coördination and strength of the muscles.

**Attention.**—The degree of attention is, in the idiot, an indication of the degree of idiocy. To a certain extent the degree of attention noted is of value in prognosis; for, if the attention can not be aroused at all, no progress in education can be made. Thus the first step in our process of education must be the employment of methods of exciting attention. The most useful are such as appeal to cutaneous sensibility, to the eye, and to the ear. But even if these are in abeyance, the other senses afford useful avenues of approach to the nervous centers. Pricking, tickling, light blows, hot and cold articles, etc., may be used to attract attention through the skin. Colored balls, brilliant pieces of cloth, a ray of light in a dark room, the magic lantern, or a spectrum—such things may be variously and patiently experimented with to fix the attention of the eye. A lead call, a bell, music, a gong, or even a pistol shot sometimes, are devices for exciting the attention of the ear. Not infrequently months of patient experiment must be traversed before we are rewarded for our labors.

**Education of the Sense of Touch.**—The methods in vogue for developing the sense of touch generally aid at the same time the coördination of muscular movements; hence in actual practice the education of the hand and touch and also of the eye proceed more or less simultaneously.

The idea of temperature is developed by plunging the hand into cold, tepid, or warm water, or by the application of bottles containing water at different temperatures.

The sense of smoothness or roughness of surface is inculcated by passing the finger-tips over a board, one-half of which is covered with velvet, the other half roughened like a grater. Pieces of stuff of varying degrees of roughness or smoothness are also made use of. The softness and hardness of objects are taught by the handling of different objects, such as hard balls or cushions.

The child is taught to button by means of two bands of cloth, one with large buttons and the other with large button-holes; to lace up a shoe, by means of a shoe with eyelets a centimeter in diameter, and



alternately hemmed with red and blue leather; to tie knots, with the aid of a pad upon which are spread strings of divers colors.

Stringing beads and buttons, sticking pins into a pin cushion covered with dotted stuff, and the use of the size-board and form-board are useful means of developing tactile sense, educating the eye, and bringing out some of the faculty of calculation.

**The Education of the Eye.**—After the physician has remedied any existing visual defects, it becomes the duty of the instructor to interest the restless and inattentive eye. As already mentioned, the attention is aroused by glittering and striking objects, and, once the gaze is captured, the latent sense may be drawn out by many devices familiar to the kindergarten and teacher. Particolored balls, variegated shapes and colors of blocks, spheres, squares, cubes, illuminated pictures, gaily-stuffed, the spectrum, the kaleidoscope—all of these play a rôle in the education of the vision of the defective pupil. The matching of ribbons, wools, or cards, and the discrimination of forms of blocks, are methods of aiding the higher development of the visual sense. The size- and form-boards already alluded to, and the use of graduated rods to be placed by the pupil in step-like rows, are excellent adjuncts. Later on come into play various games,—dominoes, ball, croquet, marbles, bean-tag, hoops, tennis, skipping, battleship and shuttlecock, quins, golf, and the like,—in the employment of all of which not only is the vision stimulated and improved, but there is a gain in manual dexterity, and an associated development of some of the psychic functions. The teacher acquires a special tact in leading the pupil to concentrate his mind upon what is being done, and in making use of the instinct of imitation, so that the child endeavours to do as the other pupils are doing or to follow the movements of the instructor.

**Education of the Sense of Hearing.**—After the physician has made sure that defective hearing is due rather to want of attention than to any of the many causes of deafness, the teacher experiments upon the sense with sounds of various kinds—gongs, bells, speech, instrumental music, and songs—and by some one of these means the ear will at last be reached and kept open until it becomes an avenue for impressions from the environment to travel to the brain for registration and the rousing of new cerebral activities. This organ in the defective is often especially alive to the influences of melody and harmony, to songs and jingles and rhymes. Music is an efficient aid in the various drills and games made use of later on in the child's mental development.

**Education of the Taste and Smell.**—While these senses have not the importance of the three just described, it is still useful to stimulate and develop them as far as possible. The child can be taught to discriminate between the simple taste sensations—salt, sweet, bitter, and sour—by means of solutions of salt, sugar, quinin, and citric acid, and between odors that are noisome and odors that are pleasant by means of tinctures of *asafoetida*, cloves, and musk, and divers perfumes. Later, he learns to distinguish flavors, and to associate what is good and useful with pleasant, and what is hurtful with noxious tastes and smells.

**Teaching to Walk.**—A course of light massage of the lower extremities, together with exercise of the joints in flexion and extension, is undertaken for the purpose of developing suppleness and strength and improving the nutrition. The child is then regularly placed in a swing constructed for the purpose, with a vertical board in front in such a position as to receive the advancing feet of the child as it moves to and fro. The impact of the feet upon the board, with the backward swing caused thereby, in the course of time gives the child a sort of pleasure, and awakes in it a sense of the dependence of its movement upon the varying pressure and impact of its feet. It is not long before the child is enabled to use its legs with considerable ease and skill in the exercise. Having attained this stage, the child is now frequently held upright on its feet and then placed between the parallel bars sustained by its arms, in which position it is induced to make efforts at walking, at first for a few minutes, but with gradual increase of the time of stay each day. Then the pupil becomes quickly ready for a wheel-chair, which is merely a modification of the principle of the parallel bars, the supports being on wheels, so that as the child walks it moves the apparatus about with it. Later on it is taught to mount and descend a stair by means of a short, stationary step-ladder. After this the gait is rapidly improved by a variety of exercises, drills, simple dances, and the like.

**Education of the Hands.**—Even though the motions of the limbs be incoördinate and without force, though the infant may be unable to do anything for itself, even to grasp an object or to oppose the thumb to the fingers, there are many methods of overcoming such defects and developing the normal power and usefulness of the hands. Among these is the employment of the parallel swinging-ladders and rings. At first the child's hands are applied to the rounds and held there by the teacher during the execution of such movements as standing, sitting down, raising the arms high above the head, and bending forward and backward, swinging to and fro, and so on.

As the pupil makes progress, the drill is carried on with great regularity and precision, accompanied by spoken commands and often with music. In this way not only are the muscles strengthened and coördinated and the use of the hands and feet perfected, but a familiarity with certain words and ideas and their association is created.

The use of blocks in building up various structures, with the subsequent pleasure of tumbling them down again, is as useful to these defectives as to normal infants.

Finger-exercises with the peg-board, or by means of picture-performing, as practised in the kindergarten, now come into play for the development of the finger movements of the hand. Some of the apparatus employed in educating the sense of touch are equally valuable for training the accurate movements of the hands.

**Teaching Habits of Cleanliness in Person and Dress.**—Idiots of every degree are slovenly, awkward, negligent, unless taught and supervised, and the lower grades are incompetent to use spoon, knife, or fork, unable to care for themselves in any way, and continually drooling, sucking their fingers, holding the mouth open, and wetting and soiling



themselves. It is of paramount importance, then, in their education to make every effort to overcome these deficiencies.

Such children as are unable or just learning to walk are placed by day in especially constructed chairs, and by night in especially prepared beds, for purposes of cleanliness, and must be watched and raised at certain hours by the nurses. It is surprising how many will, by assiduous attention, soon learn to give some signal to the care-takers of their needs, and in the end acquire control over themselves in this regard. They learn to expect the regular bath, and those who progress further become systematic in ablutions, cleansing the teeth, and all the little matters pertaining to the toilet. At the table they are taught first the use of the spoon, then of the fork, and lastly of the knife. They learn to dress themselves and to make themselves neat and tidy, and ultimately to brush and arrange their clothing, blacken their shoes, make their beds, etc. All of this instruction requires time and the utmost perseverance and patience on the part of the attendants. By it we also train the hands, the senses, and the intellect.

To close the mouth and prevent drooling, timble electrification of the orbicularis oris is employed, and the insertion of a flat piece of wood or a stick of licorice-root in the mouth is useful. The teeth need careful looking over by a dentist from time to time, and daily cleansing. Sucking of the fingers and biting of the nails can be overcome by application of doses and other bitter or disagreeable substances.

**The Teaching of Language.**—In idiots we must begin our incultivation of the uses of language according to the laws of its evolution in the normal child, first, however, correcting such defects in the ear, mouth, or vocal apparatus as are amenable to medical or surgical treatment. A child first develops its auditory word-center and then the motor speech-center. These two centers, with an association tract, are the primitive basis of language in the child. Often, in defective children, a course of gymnastic exercises of the lips, tongue, and jaw will be a necessary adjunct to the instruction, and in cases of deafness the lip-imitation method of education will require to be used.

In developing the motor speech-center the child begins by repeating the simplest linguals and labials, such as "dada," "tata," "mama," "papa," and "baba," and these first consonants should be employed in the construction of the new words to be learned.

Music is an excellent auxiliary in teaching the articulation and use of words, and Shattlenworth recommends Elliott's "National Nursery Rhymes," set to pleasing melodies, as particularly adapted for the purpose. The interest of the pupil is often best secured and sustained by the employment of objective illustrations. The naming of subjects of pictures, of persons and things about the room, of parts of the body, and the imitation of cries of animals, are means of arousing interest.

After developing the word-hearing and the motor speech-centers, the visual and writing centers will require education, and the methods in vogue are analogous to those of the kindergarten. Bourneville

recommends the use, first, of black letters twelve centimeters high; then an alphabet with the consonants in black and the vowels in red, the letters six centimeters in height; then letters of ordinary size; and, finally, the repetition, in chorus, of letters and words placed before a class. This collective exercise, in which imitation plays a great part, contributes markedly to the development of speech. Figures are employed in much the same manner, and counting is learned from some of the various apparatus already described, as well as from simpler and more interesting devices, such as the use of the fingers, shells, marbles, buttons, beads, and the abacus. The nursery game of keeping shop is especially useful for developing the ideas of number, weights, and values.

Writing and drawing are taught by means of sand-boxes, blackboard exercises, and, finally, drawing-books. The knowledge of form is best inculcated by modeling in clay, and by reproductions in clay or wood of surface drawings.

From these primary lessons it is but a step to

**Manual and Industrial Training.**—When the pupil has reached a certain stage of mental development, every effort is made to further the training to such an extent as to subserve the demands of health and utility. Methods of manual and industrial education are best furthered in institutions in which every variety of occupation commensurate with the individual needs and tastes of the pupils can be satisfactorily carried out. In most existing institutions it is true that the ideal system of care and development of defectives has not yet been attained, but the tendencies of the present time are in the right direction. The institutions of the future for all classes of dependents, for idiots, for the insane, and for the inmates of prisons and reformatories, will doubtless be modeled on the colony plan. They will be village settlements or communities wherein the chief industries will be such as relate to the housing, clothing, feeding, etc., of their inhabitants, thus bringing into existence all of the occupations which tend to utility and economical administration. The scheme is well exemplified and successfully demonstrated by the evolution of the Craig Colony for Epileptics at Syosset, N. Y. Were I called upon to draw up an outline of a plan for a colony for idiots, it would be somewhat as follows:

1. In the first place, there should be an abundance of land, at least an acre for each inhabitant. The site should be selected with due regard to fertility of soil; for agriculture, stock-raising, and gardening should afford employment for the majority of the pupils.

2. Convenience of access to managers and patients and their friends is a desideratum.

3. In the construction and arrangement of buildings the country-village idea should never be lost sight of, and the farmstead group—the cottages, villas, schools, shops, and so on—should be simple, independent, homelike, and surrounded by their own little gardens, lodges, etc., in conformity with such design.

4. So far as possible, each house should constitute a home circle, the number of members being limited to ten or fifteen.



5. An administration building, a small hospital for the sick, special villas for the infirm, bed-ridden, ineducable, and disturbed classes, a gymnasium, a library, a museum, and swimming- and rain-bath, are among the separate structures required in addition to those already mentioned.

6. The educational features of the colony will be carried on in ordinary schools, Sloyd schools, trade schools, and so on, and everything that may contribute to the furtherance of mental development should be encouraged. Thus the field study of natural history is one of the most satisfactory means of arousing the intelligence, interest, and activity of the pupils. Trees, garden produce, and flowers should be labeled with their names, botanical and zoological gardens should be established, and the collection of rocks, leaves, plants, insects, birds, etc., made a part of the system.

7. In developing the industries of the colony, such should first be instituted as will serve economical purposes. The aim should be to produce most of the food-stuffs required, to carry on domestic work, to make and mend the wearing apparel, to accomplish ordinary repairs, to construct new buildings, and to fashion the furniture.

8. The whole scheme requires to be under medical supervision, and the scientific aspects of the community thus created should be kept continually in mind. This necessitates the establishment of psychological and pathological laboratories after the most approved style.

As an instance of what species of work may be done by defectives in institutions, Bourneville's statistics of occupations at Biètré for 1897 show that there were 187 children employed in the various shops and workrooms, among them being: 10 benchmakers, 24 carpenters, 9 printers, 11 locksmiths, 51 tailors, 28 shoemakers, and 14 straw- and cane-workers. The hemiplegics work exclusively at sewing, and the blind with straw and cane. The colony plan, however, would insure a greater amount of healthier work out of doors than is possible at such a place as Biètré, and would be more remunerative to the administration.

**Moral Training and Discipline.**—Much as the inculcation of moral ideas is needed for normal children, defectives require even more attention in this respect; for in them the abrogation of higher intelligence is associated naturally with feeble inhibitive power. Thus they easily give way to the lower instincts, and are prone to acquire vicious habits of conduct and speech. In some cases the moral obliquity is so great that it constitutes the so-called moral imbecility, and little can be accomplished for their improvement. But the majority of defectives are susceptible to the influence of a good environment and moral discipline. Isolation of the teacher and of playmates and schoolmates counts for much with these. The judicious instructor and care-taker can, by firm and kindly guidance, accomplish great good in this respect, and it should always be kindly guidance, never coercion. There is, however, merit in the employment of a system of rewards and punishments adapted to the idiosyncrasies of the different pupils. A few words of encouragement or praise, or trifling compensations in the way of extra allowances of food, delicacies, recreations, or small wages, appeal dis-

timely to some; while words of disapproval, the curtailing of things pleasant to the palate, the deprivation of some anticipated pleasure, and so on, have especial influence with others. It is a good plan to distinguish the pupils for meritorious conduct and industrial accomplishments by distinctive dress, thus appealing to their ambitions. It is well to establish three or four grades to be thus distinctively recognized, for nothing is more human than the instinct to appear well to others, to be among the best-dressed. The instinctive desire of the savage for ornament is no stronger than that of the most civilized being for good clothes. The mentally feeble are no strangers to this feeling, and their good conduct can be enhanced and maintained by promotion to a better clothed division, and their shortcomings well punished by reduction to a lower rank. Corporal punishment is both necessary and useful in extreme cases with vicious tendencies, but should be a last resort even here.

By the means just described, and by other devices that will suggest themselves to the wise and careful person whom we suppose to be intrusted with their care, these unfortunates may be taught obedience, perseverance, responsibility, and regard for the rights of others, and be imbued with some knowledge of the great laws of justice, beauty, goodness, and religion which rule the ideal world of humankind.

**Physical Culture.**—The tendency to incorrectness of posture and bearing, the great lack of strength and grace, among idiots, must be overcome by systematic education of the muscles. There should be courses of gymnastic exercises and drills, with song and instrumental accompaniments. The drills may be made with rods, light dumbbells, etc. Military drill is excellent for both girls and boys. Dancing is beneficial to both mind and body. Bourneville has introduced fencing at Biotre, but does not speak of it with enthusiasm.

**The Medical and Surgical Treatment of Idiocy.**—At one time craniotomy had considerable vogue as a measure in the treatment of microcephalic idiocy, under the idea that the brain was hindered in development by premature synostoses of the skull bones. Investigation showed, however, that the sutures are normal in microcephalic skulls and not synostosed, and surgical experience finally demonstrated that nothing was accomplished by the operation. It has been hoped to do some good by surgical intervention in chronic hydrocephalus, but thus far the results have not justified any procedure of this kind. In idiocy due to trauma of the head trephining for decompression purposes (the *vental operation* of Kocher or decompression operation of Cushing) should be carried out, even in cases of long standing.

Myxedematous idiocy should, of course, be treated by the administration of thyroid extract over long periods of time, and if undertaken early enough, offers hope of a permanent cure.

**Hydrotherapy.**—The rain-bath is nowadays considered a necessary adjunct to all public institutions, because of expedition in its use and perfect cleanliness. Such baths should be the daily morning rule of defectives. The skin is kept in a hygienic state, the circulation is stimulated, and general nutrition is improved by the morning bath. In



lethargic or apathetic states the cold spinal douche is beneficial, while in very restless patients the prolonged warm bath and wet-packs at night often materially aid in overcoming the condition.

**Clothing.**—One of the noteworthy stigmas of degeneration common to all classes of idiocy is a diminished resistance to external influences and diseases. They catch cold easily. Tuberculosis and other lung disorders account for nearly seventy-five per cent. of the mortality among them. Diarrheas are common. Hence it is important that, among other things, considerable attention should be given to clothing. Woolen undergarments of warm and light texture should be the rule. The outer clothing should be light, durable, neat, of prevailing cuts and styles, and none of the clothing should in any way impede or restrict the free motions of the limbs and trunk.

**Food.**—The dietary for this class of defectives should, in my opinion, closely approximate that of epileptics—i. e., it should be chiefly vegetable, with the free use of milk and eggs, and meat but once daily. Simplicity of food and simple cooking are essential. The dietary need not be so elaborate as, for instance, in hospitals or asylums, where acute disorders are commonly treated, and where the percentage of cure is expected to be large. Idiots are apt to overeat, and hence the chief requisite is to regulate the *per capita* allowance to just the amount necessary to maintain a robust state of physical health. Overeating is probably responsible for much of the diarrhea commonly observed among these cases.

**General Bodily Health.**—Very common is a condition of general debility, which must be met by appropriate tonics, nutritive foods, special baths, massage, and regular exercise. The great mortality from tuberculosis should lead the physician to a regular examination of the viscera for symptoms of that disorder. When discovered, the usual precautions should be taken to isolate the patient and to build up the constitution in every way. Parasitic and nervous skin diseases will often need attention. The prevailing mucous diarrheas are treated by the usual remedies and by careful regulation of the kind and amount of food. Owing to feebleness of constitution and diminished resistance to diseases, especial danger attaches to acute infectious fevers in idiots.

**Masturbation.**—The prevalence of this pernicious habit among all classes of idiots is only too pronounced. In the lowest grades it is uncommon, but among the imbeciles and feeble-minded it is one of the most intractable of conditions. There are few agents and devices which have not been tried, and usually vainly, to prevent the practice. It is only rarely that vesication of the genitals, punishment, mechanical restraint of the limbs, and sedative drugs have any effect in the treatment of defectives. Indeed, they might usually as well be left untried. There have been very few experiments of the method of cure by castration, for, naturally, professional opinion is too conservative to undertake, without long and careful deliberation, so radical a remedy. I know of but one institution where castration has been apparently adopted as a part of the regular system of cure and treatment. The superintendent

of the Winfield, Kansas, Asylum for Idiots has had between twenty and thirty boys who were inveterate masturbators subjected to castration, with excellent results. Not only were their vicious habits put an end to, but there was marked physical improvement in all, and great mental improvement in most, of them. There would seem to be no reasonable objection to operative procedure in such cases, though, perhaps, it is hardly necessary to go so far as castration. Ligation of the vas deferens, or possibly section of some branch of the pudic nerve, might serve as well. At any rate, some method of this kind is well worthy of consideration, though the ultimate decision of the profession as to its utility and propriety has yet to be learned.



# INDEX.

- Abdominal reflex, 35  
in multiple cerebrospinal sclerosis, 355, 489
- Abscess of brain, 241  
diagnosis of, 245  
excysted, 245  
etiology of, 241  
hematoma in, 244  
incubation stage of, 243  
latent period of, 243  
pathology, 242  
paralytic stage of, 244  
pathological anatomy of, 242  
prognosis of, 245  
transmission stage of, 243  
symptoms of, 243  
treatment of, 245
- Accessory nerve, spinal, 189. See also Spinal accessory nerve.
- Accommodative, errors of, (importance of, 63  
in multiple sclerosis, 325
- Actinomyces in insanity, 764
- Achilles' tendon reflex, 37
- Achrochromia, 65
- Anomia of Daquinod in neuralgia, 673
- Acrocephalus, 733
- Acromegalia, 507  
course of, 513  
diagnosis of, 513  
differential diagnosis of, 513  
etiology of, 508  
forms of, 513  
mental anatomy of, 509  
prognosis of, 513  
symptoms of, 510  
treatment of, 513
- Acroparonychia, 550, 681
- Actinomyces of brain, 260
- Actions, disorders of, 758  
induced by defects of memory, 758  
by disorders of emotions, 758  
of idea-association, 759  
by sensory disorders, 758
- Active electrode, 42
- Acupuncture in sciatic neuritis, 314
- Acute anterior poliomyelitis, 388. See also Poliomyelitis.  
swelling paralytic, 376. See also Lemay's paralysis.
- Addison's blood, 529
- Adenolipomatosis, 557
- Adiaphoresis, 189
- Adipose dolorosa, 515  
diagnosis of, 517  
etiology of, 515  
pathology of, 517  
symptoms of, 515  
treatment of, 517
- Adiposity, 514
- Adonis veridica in epilepsy, 896
- Adventitious neuritis, 288
- Affective agitation, 759  
disorders, 760
- Age in etiology of insanity, 697
- Agnesia, 67
- Agitated depression, 791
- Agonizantes, 610, 736
- Agrophobia with motor aphasia, 179
- Alcohol in cerebral palsies of children, 255  
in etiology of general paresis, 821  
of insanity, 722  
in trifacial neuralgia, 674
- Alcoholic hallucinations, 723  
mania, 728  
prognosis of, 335  
paranoia, 721  
psychoses, 725
- Alcoholism, chronic, 721
- Aleus, 102
- Alienation mentis, 687
- Alimentary canal, examination of, 34
- Allophasia, 54
- Alpine scurvy, 477
- Amniotic fluid body, 258
- Amnophobia, 549
- Amnesia in insanity, 764
- American disease, 600
- Animes, 69, 177
- Annesia, 751  
retrograde, 751  
verbalis, 69
- Angetation neuritis, 292
- Ania, 177
- Anycline scotomata, 292
- Anil nitro in angina pectoris, 148  
in Raynaud's disease, 545
- Aniline iodide in mental disease, 787
- Anystonia congenita, 425
- Anystonia lateral sclerosis, 497. See also Progressive muscular atrophy, spinal.
- Anal reflex, 38  
sphincter, 38
- Analgesia, 54  
in myelitis, 398  
in syringomyelia, 492  
in tabes dorsalis, 449

- Anus, cerebral, 180  
   contused cord lesions in, 468  
   severe, 262  
 Anesthesia, 54  
   from lesions of anterior cranial nerve, 307  
     of ciliary nerve, 267  
     of external plantar nerve, 309  
     of spinal cord, 349  
   from paralysis of sciatic nerve, 308  
   hysterical, 57, 621  
     distribution of, 623  
     peculiarities of, 625  
   in mental disease, 743  
   in syringomyelia, 402  
   of larynx, 142  
   relation of, to lesions of cord, 55  
     of nerve-trunks, 55  
 Anoxypnea of brain, 240  
 Anger, 748  
 Angina pectoris, 550  
   pectoral, 547  
   hysterical, 640  
 Angioma of brain, 266  
 Angioneurotic edema, 552  
   diagnosis of, 554  
   etiology of, 552  
   prognosis of, 554  
   symptoms of, 553  
   treatment of, 554  
 Angioderotic: paroxysmal myocardial, 550  
 Anhedonia, 744  
 Anhidrosis, 27  
   spasmodic, 58  
 Anisid closing contraction, 34  
   opening contraction, 44  
 Anisodines in neuritis, 291  
   in neuromata, 294  
 Anisodines, dental, in insanity, 713  
   of appetite in insanity, 721  
   of body in insanity, 719  
   of circulation in insanity, 708  
   of ear in insanity, 714  
   of eyes in insanity, 714  
   of genital region in insanity, 719  
   of genital-urinary function in insanity, 721  
   of intellect in insanity, 721  
   of limbs in insanity, 719  
   of lips in insanity, 713  
   of motor function in insanity, 720  
   of nose in insanity, 714  
   of sensory function in insanity, 720  
   of skin in insanity, 720  
   of speech in insanity, 721  
   of tongue in insanity, 713  
 Anorexia, hysterical, 641  
 Anterior horn of cord, effect of lesions of, 332  
   median artery of cord, 342  
   roots of cord, effect of lesions of, 322  
   spinal artery, 342  
 Antiplegia in tubercular leptomeningitis, 95  
 Antiplegia in brain tumor, 270  
   in chorea, 377  
 Antiplegia in epilepsy, 666  
 Antisepsis in strain thrombosis, 238  
 Antistreptococcus serum in chorea, 577  
 Antitoxic, tetanus, in tetanus, 363  
 Anxiety, 747  
 Apathy, 748  
   from tumors in corpus callosum, 186  
 Ape hand, 300, 412  
 Aphasia, 68, 174  
   auditory, 178  
   condensed, 182  
   complete, 179  
   conduction, 181  
   examination and testing of, 69  
   graphic motor, 181  
   in cerebral hemorrhage, 212  
   in fissure of brain, 264  
   motor, 178  
     handwriting in, 179  
     varieties of, 174  
   visual, 180  
 Aphasia, production of, 182  
 Aphasia, 178  
 Aphasia in hysteria, 68  
 Aphthous, 153  
 Apoptosis, 506  
 Apomorphin in chorea, 577  
 Apoplectic state, 216  
   "stroke," 216  
 Apoplexy, hemorrhagic, 217  
 Aporeceptive dementia, 815  
 Appetite, anomalies of, 721  
 Apraxia, 59, 169, 177, 180  
   in fissure of brain, 264  
 Amblyopia, 72  
 Arc de cercle, 653  
 Argyll Robertson pupil, 54  
   in insanity, 262  
 Arcs, nerves of, combined plexus of, 362  
 Arsenic in beer, multiple neuritis from, 346, 355  
   in cerebrospinal syphilis, 562  
   in chorea, 577  
   in multiple neuritis, 335  
   in neurasthenia, 669  
 Arsenobenzol in general paresis, 815  
 Arterial lesion diseases, 197  
 Arteries, acute degeneration of, 207  
   of brain, 197  
   of spinal cord, 242  
 Arteriosclerosis, cerebral, 203  
   symptoms of, 201, 205  
   treatment of, 205  
 Arsenia, cerebral, 202  
   syphilitic cerebral, 492  
   of brain, 206  
   symptoms of, 496  
 Artery, anterior median, of cord, 342  
   spinal, 342  
 Articular muscular atrophy, 40, 426  
   diagnosis of, 428  
   etiology of, 426  
   nerve anatomy of, 427  
   pathology of, 427  
   progress of, 428  
   symptoms of, 426  
   treatment of, 428



- Arteritis, acute, in cerebral hemorrhage, 212  
     diphlococcus of Weichselbaum in, 79  
     relapse of, 10 ebers, 368  
 Arthropathies in syringomyelia, 404  
 Arthropathy, dystrophic, 40  
     tabetic, 431  
 Aschmolechthy, 719  
 Aspirin in tabes dorsalis, 463  
 Associated movements, 32  
     in infantile cerebral palsy, 230  
 Association disease, 656  
 Atasia abusa, 638  
 Asterognosis, 51  
 Athetoid bulbar paralysis, 162  
 Atkiss, bronchial, 143  
     symptoms of, 144  
     treatment of, 144  
     mononitic, 143. See also Atkiss, bronchial  
 Ataxian gait, 477  
 Ataxias for insens, 776  
 Asymmetrical palsy, 709, 712  
 Asymmetry, facial, 707  
     of skull, 701  
     physiological, 702  
 Ataxion, 18  
 Ataxia, detection of, 29, 39  
     family, 479. See also Family ataxia.  
     Friedreich's, 479. See also Family ataxia.  
     hereditary cerebellar, 470  
     in tabes dorsalis, 436  
     infantile, 845  
     hemiparetic, progressive, 429. See also Tabes dorsalis  
     progressive spastic, 464  
     static, 39  
 Ataxic gait, 436  
     hunting, 39  
     paraplegic, 464  
     cycloptic, 509  
 Ataxias of cerebral vessels, 202  
 Ataxicostosis anterior in etiology of insanity, 726  
 Athetoid movements in infantile cerebral palsy, 249  
 Atosis, 32  
     in infantile cerebral palsy, 249  
     treatment of, 255  
 Atrophy, 317  
 Atonic paraplegia, congenital, 425  
 Ataxia in sleeping sickness, 681  
 Atrophied musculature progressive spinal, 408  
 Atrophy, atrophic, muscular, 44, 425.  
     See also Atrophic muscular atrophy from lesions of spinal cord, 359  
     in facial paralysis, 126  
     in multiple neuritis, 323  
     in myelitis, 349  
     in syringomyelia, 405  
     of eighth nerve, 503  
     of optic nerve, 100  
     optic, 505  
     progressive muscular, 403. See also Progressive muscular atrophy.  
 Atrophy in etiology of insanity, 724  
     in myoclonia, 583  
 Attention, disorders of, 751  
 Attitude, importance of, 28  
     in atrophic muscular atrophy, 426  
     in paraplegia, 375  
     in scute neuritis, 311  
 Auditory aphasia, 178  
     hyperesthesia, 65, 111  
     nerve, atrophy of, 505  
     electrical testing of, 99  
     irritation of, 131  
     paralysis of, 132  
     symptoms in cerebellar disease, 191  
 Auras, epileptic, 551  
 Axial vertigo, 134  
     diagnosis of, 135  
     treatment of, 137  
 Axillobrachial radii, 704, 710  
 Axolymphoma, 562  
 Axo-intoxication in etiology of insanity, 722  
 Axonal oblique in dementia parva, 814  
 Axon ear, 716  
 Backward's foreign, 57  
     in myelitis, 323, 376  
 Basilar end in hypostomatitis, 79  
     tubercle in hypostomatitis, 79  
     in tubercular meningitis, 92  
 Baring's labyrinthine test, 146  
 Basal ganglia, functions and lesions of, 157  
 Basche's disease, 528. See also Erythema basche ganglia  
 Basilar meningitis, 91. See also Leptomeningitis, tubercular  
 Bason, 205  
 Batteries for electrical testing, 41  
 Beard's disease, 600  
 Behism in cerebral hemorrhage, 212  
 Beer, arsenic in, multiple scurvy from, 136, 325  
 Behism in epilepsy, 609  
     in encephaloid palsy, 517  
     in nocturnal convulsions, 609  
 Bell's palsy, 121  
 Bence, 382  
 Bence-Wiley syndrome, 158  
 Benedikt's epilepsy, 705, 706  
 Ben-Dani, 316, 320  
 Betanin, 669  
     in auto-intoxication, 783  
     in epilepsy, 567  
     in myelitis, 372  
 Birkhead of memory in anterior myelitis, 397  
     in Landry's paralysis, 380  
 Bir's treatment in writer's cramp, 720  
 Bitumensol, 704, 710  
     number, 704, 710  
 Blackie ear, 715  
 Biphosphorus as a symptom, 62  
 Blackness, functional, 105  
     time, 107

- Blood-serum examination in cerebrospinal syphilis, 502
- Body, anomalies of, 719
- Bones, testing sensibility of, 54
- Brachial plexus, lesions of, 503
- neuritis of, 394. See also Neuritis
- Brachycephalic head, 702
- Bradycardia, 146
- Brain, abscess of, 241. See also Abscess of brain.
- anatomy of, 260
- arterial supply of, 197
- carcinoma of, 258
- contusion of, 728, 729
- cysts of, 259
- disease, path in, 62
- glioma of, 257
- inflammation of, 238
- lesions of, destructive, 192
- general considerations of, 192
- hematoma in, 194
- irritative, 192
- mental disturbances in, 194
- vertigo in, 194
- vomiting in, 194
- sarcoma of, 257
- softening of, 222
- syphilis of, 502. See also Syphilis, cerebral.
- tubercle of, 256
- tumors of, 256. See also Tumors of brain.
- syphilitic, 259
- Brandy in mania, 708
- Break of current, 42
- Bregmatolambdoid arc, 704, 707
- Bright's disease, as predisposing to nervous disease, 19
- Brodie's points, 622
- Broad in cerebral softening, 230
- in family periodic paralysis, 392
- of potassium in family periodic paralysis, 392
- in tubercular leptomeningitis of children, 95
- Bronchi in brain tumor, 226
- in epilepsy, 666
- of infantile cerebral palsy, 255
- in encephalitic gait, 527
- in mental disease, 786
- in multiple neuritis, 336
- in myoclonic epilepsy, 657
- in senile dementia, 922
- in tetanus, 563
- Bronchial asthma, 142. See also Asthma.
- Brown-Séquard paralysis, cord lesion in, 56, 57
- Bruit in intracranial aneurysm, 195
- Bulbar paralysis, acute, 162
- asthenic, 162
- progressive, 157. See also Pseudo-bulbar inferior sclerosis
- Bulbo cavernosus reflex, 448
- Bulimia, 744
- CACHEXIA, combined cord lesions in, 468
- stranguria, 519
- Caffein in brain tumor, 270
- in family periodic paralysis, 392
- in migraine, 665
- Cagot ear, 717
- Callosal disease, 380
- etiology of, 380
- marked anatomy of, 381
- periphasis of, 382
- symptoms of, 381
- treatment of, 382
- Calabar bean in tetanus, 563
- Calipers, 705, 706
- Callosal in hydrocephalus, 274
- in leptomeningitis, 57
- in tubercular leptomeningitis, 95
- Camphor in bronchial asthma, 145
- Cannabis indica in etiology of insanity, 724
- in migraine, 665
- in Parkinson's disease, 588
- in torticollis, 150
- Carbonic acid in tetanus, 563
- Carcinoma in etiology of insanity, 726
- of brain, 258
- Cardiac branches of vagus, diseases of, 145
- palpitation, 146
- Cardiopathy, relation of, to clarea, 568
- Cardiomyoid cephalothalamus, 528
- Carriers in poliomyelitis, 337
- Cashbook, value of 70
- Cataleptic form of dactylitis parva, 817
- Cathodal closing contraction, 44
- tetanus, 44
- opening contraction, 44
- Cauda equina, lesions of, 560
- Center for elbow movements, 169
- for face movements, 169
- for finger movements, 169
- for hearing, 171
- for lower extremities, 169
- face movements, 169
- for platysma, 168
- for shoulder movements, 169
- for smell, 171
- for speech, 170, 174
- for taste, 171
- for tongue movements, 169
- for trunkal movements, 169
- for vision, 170
- for wrist movements, 169
- Centers, cortical, 167
- relation of body to, 169
- for word memories, 174
- motor, of cerebral cortex, 167
- Central canal of cord, effect of lesions of, 332
- myelitis, 366
- scoliosis, 103
- Cephalalgia, hysterical, 629
- Cephalic index, 702
- tetanus, 562
- Cerebellar hemorrhage, 218
- Cerebellopontine angle, tumors in, symptoms presented by, 191
- Cerebellopontine gait, 484
- Cerebellum, function of, 189



- Cerebellum, lesions of, headache in, 191**  
 symptoms of, 190, 191  
 vertigo in, 190
- Cerebral anemia, 199**  
 diagnosis of, 200  
 etiology of, 199  
 symptoms of, 199  
 treatment of, 200
- arteriosclerosis, 203**  
 arteritis, 202  
 cystic, 200, 492
- cortex, cells of, 166**  
 latent lesions of, 171  
 lesions of, effects of, on sensation, 52  
 localization in, 165. *See also Local-  
 ization; cerebral*  
 motor centers of, 167  
 unknown functions of, 171
- hemorrhage, 202. *See also Hemor-  
 rhage***
- hypertension, 200**  
 diagnosis of, 201  
 etiology of, 201  
 symptoms of, 201
- lesions in tabes dorsalis, 235**
- messages, anatomical considerations,  
 72**  
 diseases of, 72
- meningitis, syphilitic, 492**
- palsies of children, 246**  
 causes attending birth, 246  
 classification of, 248  
 diagnosis of, 254  
 diplegic cases, 251  
 epileptic attacks in, 254  
 etiology of, 246  
 hemiplegic cases, 248  
 morbid anatomy of, 244  
 postnatal causes of, 247  
 prenatal causes of, 246  
 prognosis of, 254  
 symptoms of, 248  
 treatment of, 255
- perinatal, 202**
- atrophy, 231**
- softening, 222**  
 abrupt onset in, 225  
 course of, 225  
 diagnosis of, 227  
 differential diagnosis of, 228  
 etiology of, 224  
 location of, 224  
 paralytic state in, 226  
 pathological anatomy of, 221  
 prognosis of, 229  
 progressive form in, 226  
 rel., 225
- sensory disturbances in, 227**  
 symptoms of, 225  
 treatment of, 230  
 white, 225  
 yellow, 225
- syphilis, 492. *See also Syphilis, cere-  
 bral***
- trini, 231**
- white matter, function of, in localiza-  
 tion, 196**
- Cerebrum, 238**  
 acute, localized, 238, 240  
 etiology of, 238  
 pathological anatomy of, 239  
 symptoms of, 239
- chronic, 241**  
 from cerebral hemorrhage, 242  
 syphilitic, 492
- Cerebrum, 238**  
 Cerebrospinal meningitis, 78. *See also*  
*Leptomeningitis*
- Cervical ribs in neuritis of brachial  
 plexus, 305**
- Charcot-Marie disease, 424**
- Charcot's disease, 407, 414**  
 joints, 40, 422
- Chiasm, optic lesion of, 390, 394**
- Children, cerebral palsies of, 246. *See*  
*also Cerebral palsies of children***
- Chiasm, 393**
- Child, hydrate in chorea, 376**  
 in insensibility, 677  
 in mental disease, 787  
 in multiple neuritis, 336  
 in status epilepticus, 867  
 in tetanus, 563
- Child of iron in anterior poliomyelitis,  
 397**  
 fracture of, in Landry's paralysis, 280
- Chloroform in angina pectoris, 148**  
 in bronchial asthma, 145  
 in tetanus, 563
- Cholostomatoma of brain, 200**
- Chorea, 366**  
 adult hereditary, 578  
 rubric disorders in, 573  
 chronic, 578  
 complications of, 577  
 corpuscles, 569  
 course of, 574  
 diagnosis of, 576  
 electric, 576, 581  
 etiology of, 566  
 family, 578  
 fibrillary, 580  
 forms of, 575  
 general state in, 574  
 glycolysis, 575  
 grave, 575  
 handwriting in, 572  
 Huntington's, 578  
 limp, 575  
 mental symptoms of, 571  
 minor, 566  
 morbid anatomy of, 569  
 motor symptoms of, 571  
 of pregnancy, 575  
 of Sydenham, 566  
 of the aged, 578  
 paralytic, 575  
 pathogenesis of, 568  
 prognosis of, 576  
 relations of rheumatism to, 568  
 scale, 580  
 symptoms of, 570  
 treatment of, 576

- Choric status, 575  
 Chorioid movements in infantile cerebral palsy, 249  
 Chorioid in leptomeningitis, 82  
 Chronic delusional insanity, 836. See also *Paranoia*.  
 Chvostek's sign, 545  
 Clancy reflex, 33  
 Cerebral insularity, 805  
   course of, 807  
   etiology of, 804  
   maternal period of, 805  
   melancholy period of, 804  
   prognosis of, 792  
   symptomatology of, 804  
   treatment of, 808  
   varieties of, 807  
 Cerebrum apparatus, examination of, 35  
 Cerebral cortex, lesions of, 297  
 Cerebrum intermitto, 533  
 Claustrophobia, 610, 736  
 Cleft-palate, 719  
 Clonic convulsions, 32  
   spasm, 34  
 Clonic, urticar, 37  
   hairs, 37  
   nails, 37  
   arms, 35  
 Coccyx in etiology of insanity, 724  
   in multiple neuritis, 334  
   in myeloma, 581  
   in neuritis, 672  
   in urticar neuritis, 315  
   inflammation in herpes zoster, 339  
 Codon in epilepsy, 566  
   in mental disease, 796  
 Cog-wheel sign in paralysis agitans, 588  
 Collod of silver, Crook's, for hypotonia, 216, 88  
 Colloiditis, 65  
 Coma, phlegm, from tumors in corpus callosum, 186  
 Combined aphasia, 182  
   cord lesions in stenosis and rachis, 468  
   palsies of nerves of arm, 362  
   sclerosis of the spinal cord, 464  
   course of, 466  
   diagnosis of, 467  
   etiology of, 464  
   anatomical anatomy of, 465  
   prognosis of, 467  
   symptoms of, 466  
   treatment of, 467  
   tabes, 464  
 Coma of brain, 728, 729  
   of spine, 666  
 Coma aphasia, 183  
 Congenital atonic pseudoparalysis, 425  
   myeloma, 621  
   paraneuritis, 780  
 Contractility in facial paralysis, 126  
 Congruity of parents as a predisposing cause of nervous disease, 18  
 Constant current, test of, in health, 42  
 Continuous epilepsy, 657  
 Contraction, anastomosing, 44  
   opening, 44  
   callosal closing, 44  
   opening, 44  
   front-top, 37  
   pseudoclosed, 37  
 Contracture, 31  
 Contractures from cord lesions, 347  
   in infantile cerebral palsy, 249, 250  
   in multiple neuritis, 322  
   of hysteria, 636  
 Convulsions, 32  
   clonic, 32  
   general, 32  
   in cerebral palsy of children, 249  
   in leptomeningitis, 82  
   in tubercular leptomeningitis, 95  
   in tumor of brain, 262  
   Jacksonian, 32  
   local, 32  
   tonic, 32  
 Coprolalia, 614, 757, 817  
 Cord lesions, combined, in stenosis and rachis, 468  
   cross, table of symptoms in, 335-338  
   horizontal localization of, 351  
   indeterminate, 362  
   motor symptoms of, 347  
   paralysis from, 347  
   reflexes in, 349  
   sensory symptoms of, 349  
   trophic conditions in, 350  
   vasomotor disturbance in, 350  
   visceral symptoms of, 350  
   substance, traumatic lesions of, 362  
 Corona radiata, function of, 184  
 Corpus quadrigemum, function of, 187  
   lesions of, 184  
   symptoms of, 187  
   stimuli, function of, 187  
 Corpus callosum, function of, 186  
   lesions in coxothalamic palsy, 529  
 Cortex, cerebral. See *Cerebral cortex*.  
 Cortical localization, motor, 167  
   sensory, 170  
   paralysis in insanity, 764  
 Cough, nervous, 145  
 Counterstimulation in combined sclerosis of cord, 467  
   in Landry's paralysis, 380  
   in neuritis, 291  
   in urticar neuritis, 314  
   in spinal leptomeningitis, 282  
   in syringomyelia, 406  
   in tabes dorsalis, 461  
 Cowling, hysterical, 638  
 Cramp, 31  
   arterial, 588, 761  
 Crisis progressus, 765  
 Cranial aneurysm, 701  
   nerves, affections of, in leptomeningitis, 82  
   lesions of, in tabes dorsalis, 425  
   multiple paralytic of, 154  
   syphilitic lesions of, 482  
 Craniotomy in microcephalic alcy, 916  
 Craniocerebral topography, 171



- Cranioethelial measurements, 704  
 table of, 707
- Cranium, anomalies of, 700  
 deformations of, 704  
 measurement of, 704  
 physiological asymmetry of, 702
- Cranio, 829
- Crode's coil of silver circuit for leptomeningitis, 88
- Cremasteric reflex, 38
- Cristian, 522
- Crozier, 521
- Cross, gastric, 446  
 laryngeal, 449  
 nephritic, 448  
 pharyngeal, 449  
 tabetic, 446  
 visceral, 446
- Cross-sections of cord, table of symptoms in, 353-359
- Cross comb, function of, 188  
 results of lesions of, 188
- Cranial nerve, anterior, lesions of, 307
- Cure in tetanus, 365
- Current, break of, 42  
 make of, 42
- Cushing's decompression operation in idiocy, 910
- Cutaneous areas, relation of, to spinal-cord segments, 56, 59  
 distribution of nerves, 52, 53  
 sensibility, electrical testing of, 49
- Cyctologia, 68
- Cysts of brain, 229  
 subdural, of spinal cord, 384
- Dactyloscopy, 65
- Diamerostomus, 900
- Darwin ear, 716
- Deafness, diagnosis of cause of, 153  
 nervous, 153  
 treatment of, 153
- Debilitating diseases as predisposing to nervous disease, 18
- Decompression operation for idiocy, 910
- Decubiti from trophic disturbance, 40
- Delirium from cerebral pulse of children, 249
- Depressivity, signals of, 21
- Degeneration, progressive leucotical, 187  
 reason of, 46  
 secondary, of spinal nerve, 285  
 signals of, 499
- Delire de négation généralisé, 755
- Delirium insidiosum, 720  
 in anterior poliomyelitis, 323  
 in leptomeningitis, 81  
 in multiple sclerosis, 728  
 transient, 724
- Delusion of grandeur, 755  
 of negation, 755  
 of preservation, 754
- Delusory, 753  
 effect of, on actions, 761  
 in paralytic dementia, 828
- Delusory, systematization of, 759
- Dementia, 819  
 asperceptive, 815  
 definition of, 823  
 epileptic, 822  
 paralytic, 827, 829, 822  
 definition of, 822  
 diagnosis of, 831  
 duration and prognosis of, 829  
 etiology of, 823  
 pathological anatomy of, 833  
 symptomatology of, 824  
 treatment of, 834
- Jerome, catatonic form of, 814, 817  
 classification of, 811  
 diagnosis of, 819  
 etiology of, 811  
 hebephrenic form of, 826  
 paralytic type of, 815  
 prognosis of, 818  
 simplex, 816  
 symptoms of, 817  
 treatment of, 819
- secondary, 819  
 segmental, 815  
 senile, 818, 820  
 course and prognosis of, 821  
 diagnosis of, 823  
 etiology of, 820  
 pathological anatomy of, 821  
 symptomatology of, 820  
 treatment of, 822
- senile, juvenile, 820
- Depression, 746  
 agitated, 701
- Derog's disease, 325
- Dermatoglyphs, 477
- Dermatograms in hysteria, 642
- Destructive brain lesions, 192
- Destructiveness in insane, management of, 700
- Diabetes a predisposing cause to nervous disease, 19
- Diagnosis in neurology, importance and difficulty of, 17
- Diet in insanity, 782  
 in leptomeningitis, 88
- Diffuse symptoms, 794
- Digitalis in epilepsy, 699  
 in morbidus puer, 517
- Digit scutis, 538
- Diphtheric paralysis, 729
- Diplegia, 29  
 in infantile cerebral palsy, 251
- Diplococcus interlobularis meningitidis in leptomeningitis, 78  
 of Weichselbaum in leptomeningitis, 79  
 variety of meningitis, 90  
 course of, 90  
 diagnosis of, 90  
 etiology of, 90  
 prognosis of, 90  
 symptoms of, 90  
 treatment of, 90
- Diplopia, 62  
 in ocular paralysis, 110  
 monocular, 63

- Diplopia test, 111  
 Disease, electrical tests in, 45  
     in etiology of insanity, 725  
 Disorders of action, 758  
     of idea-associations, 759  
 Disorientation, 759  
 Disseminated myelitis, 366  
     chronic, 451. *See also Multiple cere-*  
*brospinal sclerosis.*  
 Distractibility in mania, 794  
 Disease, trophic disturbances from, 49  
 Driver's palsy or paralysis, 589  
     etiology of, 589  
     nerve anatomy of, 581  
     symptoms of, 582  
     treatment of, 582  
 Division of nerves, 285  
 Dolichocephalic heads, 702  
 Dome-shaped palate, 599, 711  
 Dreams, 21, 678, 774  
     Fernal's views, 775  
 Drop-foot in multiple neuritis, 319, 329  
 Drop-wrist in multiple neuritis, 321  
     in neurodegenerative disease, 330  
 Drowsiness, pathological, 721  
 Duboué's disease, 581  
 Duboué in epilepsy, 567  
     in mania, 708  
     in mental disease, 786  
 Duchenne-Aitkin's disease, 407, 414  
 Dura mater, 72  
     hematomas of, 74  
     inflammation of, 74. *See also*  
*Pachymeningitis.*  
 Dynamometer, kindle, (cf. Mathieu), 28  
 Dysconjugia, 66  
 Dysorthesia, 55  
 Dyspepsia, nervous, 149  
 Dystrophic arthropathy, 60  
 Dystrophy, genital, 514  
 Ear, movement of, 714  
     Atlee, 715  
     Blairville, 711  
     Cugat, 717  
     Harris, 716  
     Jesse, 702  
     Mend, 715, 716  
     Stahl, No. 1, 715, 716  
     No. 2, 716  
     Widernoth, 708, 718  
 Echolalia, 68, 170, 770, 817  
 Eclampsia, 759, 817  
 Ectasy, hysterical, 635  
 Electrodiagnosis, 739  
 Ectrodesia, 710  
 Edema, acute prescribed, 552  
     angioneurotic, 552  
     chronic hereditary, 560  
 Education of idiots, 909  
 Eighth cranial nerve, anatomical con-  
     siderations of, 130  
     diseases of, 130  
     nerves, auditory branch of. *See Audi-*  
*tory nerve.*  
     vestibular branch of. *See Vesti-*  
*bular nerve.*  
 Elbow, center for movements of, 169  
 Elbow-jerk, 34  
 Electrical shock, 576, 581  
     conditions, 41  
     examination in polioencephalitis in-  
     terior chronic, 160  
     testing, arrangement of electrodes in,  
     42  
     batteries for, 41  
     for motor areas of brain, 49  
     in disease, 45  
     of cutaneous sensibility, 49  
     of hearing, 43  
     of taste, 49  
     of vision, 49  
     tests in health, 42  
 Electricity in anterior poliomyelitis, 286,  
     309  
     in encephalic palsy, 537  
     in facial paralysis, 129  
     in family ataxia, 478  
     in infantile cerebral palsy, 253  
     in intermittent hiccuping, 551  
     in Latzky's paralysis, 589  
     in laryngeal paralysis, 142  
     in multiple neuritis, 336  
     in myelitis, 373  
     in neurasthenia, 609  
     in neuritis, 294  
     of brachial plexus, 306  
     in psychasthenia, 613  
     in Raynaud's disease, 549  
     in white scurvy, 114  
     in spinal progressive muscular atrophy,  
     445  
 Electrode, active, 42  
     indifferent, 42  
 Electrodes, arrangement of, in electrical  
     testing, 42  
 Electrophoresis in insanity, 785  
 Epilepsia, cerebral, 222  
 Emotional indifference in dementia pro-  
     prie, 813, 814  
 Eructations, disorders of, actions induced  
     by, 738  
     galvanometer test for, 773  
     in dementia propro, 813  
     with galvanometer test for, 772  
 Empirical greatest height of head, 704  
 Encephalitis in cerebellar disease, 190  
 Encephalitis, acute hemorrhagic, 240  
     diagnosis of, 241  
     etiology of, 240  
     nerve anatomy of, 240  
     symptoms of, 240  
     treatment of, 241  
     chronic, 241  
     epidemic, 241  
     in cerebral softening, 223  
     lethal, 241  
 Encephalomalacia. *See Cerebral infar-*  
*ction.*  
 Encephaloma deformans of cerebral ves-  
     sels, 202  
 Exaltis artemisi, 523  
 Excess, nocturnal, 679  
 Exertive sensibility, 55



- Epidemic encephalitis, 241  
paralytic, 368
- Epidual method of injecting salt solution, 314
- Epigastric reflex, 45
- Epilepsia partialis continua, 857  
protracta, 655  
tarda, 822
- Epilepsy, 647  
attack of, 654. *See also Epileptic attack*  
stage of attacks of, 651  
complete convulsion of, 652  
continua, 652  
diagnosis of, 657  
differential, 658  
duration in, 655  
etiology of, 648  
general state in, 657  
in etiology of insanity, 727  
initial forms of, 648  
symptoms, 656  
sustained, 654  
pathology of, 649  
postepileptical phenomena of, 655  
prodromes of attacks of, 654  
progress of, 658  
status epilepticus in, 654  
treatment of, 659  
unstable fits, 654  
vertiginous attacks in, 654
- Epileptic attack, 650  
acute of, 650  
cerebral, 652  
motor, 651  
psychic, 651  
sensory, 651  
clonic period of, 651  
epileptic, 652  
in infants cerebral palsy, 251  
incomplete, 654  
period of motor, 653  
psychic equivalents of, 655  
tonic stage of, 652  
vertiginous, 654
- dementia, 862
- insanity, 860  
acute transitory, 863  
chronic, 864  
diet in, 867  
moral treatment of, 865  
treatment of, 864
- Epilepsy, psychio-degenerative of, 862
- Epidemic after middle life, 263
- Erk's paralysis, 424  
phenomenon, 545
- Ergot in acute spinal leptomeningitis, 281  
in catarrh disease, 582  
in spinal meningitis leucomeningitis, 284  
in tuberculous, 463
- Ergotism, 477
- Erythraemia in leptomeningitis, 54
- Erythrocalcaemia, 520
- Esterin in myeloma, 581
- Esmarch's bandage in Raynaud's disease, 549
- Euphorbia, 139
- Esthesiometer, 31
- Esterin in tetanus, 563
- Ewald's labyrinthine test, 136
- Exalgin in dyslexia, 377
- Exaltation, 747
- Examination in insanity, methods, 765, 770  
in various diseases, 17
- Exophthalmic goiter, 528  
diagnosis in, 531  
radiocervical symptoms of, 531  
course and progress of, 536  
diagnosis of, 536  
digestive disturbances in, 535  
etiology of, 528  
genital disturbances in, 536  
goiter in, 531  
hemorrhage in, 534  
hereditary disturbances in, 534  
normal anatomy of, 530  
motor symptoms of, 535  
muscles in, 530  
ocular symptoms of, 532  
respiratory changes in, 535  
secretory symptoms of, 534  
skin in, 535  
symptoms of, 530  
tables of, 536  
treatment of, 537  
vascular symptoms of, 534
- Exophthalmos, 528
- Extension symptoms, 192
- Extrajugal hemorrhage, 76
- Eyes, anomalies of, 714
- Exostosis, influence of, 61
- Face, center for movements of, 199
- Facial asymmetry, 707  
expression in insanity, 768-769  
hemiatrophy from disease of trifacial nerve, 543  
length, 704, 707  
nerve, 121  
anatomical considerations of, 121  
paralysis of, 121. *See also Facial paralysis*  
spasmodic affections of, 122  
paralysis, 125  
alternating, 126  
course of, 126  
diagnosis of, 127  
double, 126  
infant, 129  
peripheral, etiology of, 123  
progress of, 126  
supranuclear, 129  
symptoms of, 124  
treatment of, 129  
types, 122
- Facioscapulothoracic form of muscular atrophy, 424
- Falling sickness, 647
- Family stigma, 470  
course of, 475  
diagnosis of, 475  
etiology of, 471

- Family ataxia, Friedreich's form of, 473  
 Marie's form of, 475  
 morbid anatomy of, 472  
 prognosis of, 476  
 symptoms of, 473  
 treatment of, 476  
 varieties of, 475  
 chorea, 378  
 history in insanity, 767  
 myositis, 388  
 periodic paralysis, 591  
 tremor, 388
- Faradic current, test with, in health, 42  
 Fatigue neuroses, 593  
 Fever, hysterical, 642  
 Fibillar neuromata, 292  
 Fibillary chorea, 346  
 Insane, 30  
 Fibrosis of brain, 309  
 Fibular point, 311  
 Field of vision, 84. *See also* Visual field  
 Fifth nerve. *See* Trigeminal nerve  
 Fingers, center for movements of, 199  
 First cranial nerve. *See* Olfactory nerve  
 Fixed point, 64  
 Flat-foot in intermittent limping, 551  
 in talus, 432  
 Flat-headness, 503  
 Flattened pulse, 709, 711  
 Flax, 66  
 Flexibility cortex, 752, 759  
 Fluor's center in acute spinal lepto-  
 meningitis, 281  
 in dyslexia variety of meningitis,  
 89  
 in leptomeningitis, 88  
 Flight of ideas in dementia praecox, 831  
 Hydiometer in leptomeningitis, 88  
 Foerster's method of division of nerve-  
 roots in cerebral lesions of children, 255  
 Folic, 687  
 a clois, 734  
 du clois, 737  
 hypose, 738  
 simultane, 733  
 Foot-drops, 32  
 Foot-drop in multiple neuritis, 319, 320  
 Forced movements in cerebellar disease,  
 181  
 in disease of labyrinth, 66  
 position in cerebellar disease, 191  
 Forgetfulness in multiple neuritis, 326  
 Fomae frustes in multiple sclerosis, 488  
 Fourth nerve, anatomical considerations  
 of, 108  
 effect of division of, 199  
 Fowler's solution in chorea, 377  
 Freud's views on disease, 775  
 Friedmann sign, 739  
 Friedreich's ataxia or disease, 470. *See*  
 also Family ataxia  
 Friedlöh's dystrophie adipogenitale,  
 597  
 Frontal lobe, 173  
 Front-clip-contraction, 37  
 Functional blindness, 107  
 nervous diseases, 369
- Functional stigmata of degeneracy, 22  
 Furoe epileptics, 748
- Garr, importance of, in diagnosis, 28  
 in family ataxia, 473  
 in idiopathic muscular atrophy, 421  
 in multiple neuritis, 319  
 sclerotic, 484  
 in Parkinson's disease, 385  
 in static neuritis, 311  
 paralytic, 374
- Galvanic current, test of muscle by, in  
 health, 42  
 Galvanometer test for reactions, 773  
 Ganglion neuroma, 292  
 Gastrology, 148  
 Gastric branches of vagus, diseases of,  
 148  
 crises, 446  
 Gastrolysis, 148  
 Gastro-intestinal disorders in etiology of  
 insanity, 726  
 General convulsions, 32  
 paralysis of the trachea, 822. *See also*  
*Paralytic dysentery*  
 paros, 457, 822. *See also* *Paralytic*  
*dysentery*  
 Geniculate bodies, lesion of, 164  
 Genital disease in etiology of insanity, 726  
 dystrophy, 514  
 organs, anomalies of, 729  
 Genito-urinary function, anomalies of,  
 721  
 tract, examination of, 26  
 Genit evelling, 552  
 articular, 552  
 Gignation, 553  
 Gentle sensation in talus dorsalis, 446  
 Glabellar point, 172  
 Glabellar neuritis, 506  
 Glioma of brain, 257  
 of spinal cord, 285  
 Globus, 159  
 hysterical, 639  
 Glossopharyngeal nerve, anatomical con-  
 siderations of, 128  
 diseases of, 138  
 Gluteal nerve, lesions of, 707  
 point, 311  
 Glycosuria in acromegalia, 537  
 in insanity, 764  
 Gutter, ophthalmic, 528. *See also*  
*Erythematous gutter*  
 Guttie pulse, 709, 710  
 Gowers' rule in testing diplopia, 111  
 Gracilis, definition of, 755  
 Graphic motor aphasia, 181  
 Graphopoint, 693  
 Graves' disease, 528. *See also* *Eryth-*  
*ematous gutter*  
 Gray matter of cord, lesions of, 288
- Haar-quora, 644  
 Habit, importance of, in diagnosis, 29  
 Habit, investigation of, 19



- Hallucinations**, 728  
conditions in which they occur, 741  
effect of, on actions, 738  
examination for, 743  
of memory, 739  
origin of, 740
- Hallucinatory agitation**, 739  
confusion, 741  
stupor, 741
- Hallucinosis, alcoholic**, *see*, 723  
traumatic, 728
- Hammer toe**, 339  
in family atasia, 474
- Hand-dynamometer of Mathews**, 28
- Hand-writing**, examination of, 69  
in dience, 372  
in motor aphasia, 179  
in parietal, 825
- Healing**, 713
- Headache in etiology of insanity**, 724
- Head injuries in etiology of insanity**, 728  
retraction of, in cerebellar disease, 191  
tetanus, 362
- Headache as cerebral symptom**, 194  
in brain disease, 244  
lesions, 194  
tumors, 232  
in cerebral syphilis, 194  
in cerebellar syphilis, 297  
in epilepsy, 632  
in leptomenigitis, 81  
in lesions of cerebellum, 194  
in migraine, 661  
in neurasthenia, 612  
in pituitary hemorrhage, 78  
in tubercular leptomenigitis, 92  
meningitis, 92  
in tumor of brain, 262  
lead cap, 632  
occipital, in cerebellar disease, 191  
rock, 661
- Health, electrical tests in**, 42
- Hearing, affections of**, 134  
in leptomenigitis, 81  
center for, 171  
electrical testing of, 41  
hallucinations of, 739  
illusions of, 712  
in facial paralysis, 126  
in idiocy, 579  
in mental patients, 132  
testing of, 45
- Heart disease in etiology of insanity**, 729
- Heterokinetic form of dementia praecox**, 816  
insanity, 731
- Hibetude from tumors in corpus callosum**, 186
- Hiboklophrenia**, 816
- Hick-Jensen reflex**, 37
- Hime-Melin's disease**, 388
- Hematomas cerebri**, 262  
of dura mater, 74
- Hematomyelia**, 362  
diagnosis of, 364  
etiology of, 362  
anatomical anatomy of, 363
- Hematomyelia, prognosis of**, 364  
symptoms of, 363  
lesions of, 364
- Hemianesthesia in cerebral hemorrhage**, 212
- Hemianopsia pupillary reaction**, 103
- Hemianopsia, explanation of** (production of), 101  
from cerebral hemorrhage, 212  
in tumor of brain, 264  
lateral hemianopsia, explanation of (production of), 102
- Hemiparesis**, facial, from disease of trifacial nerve, 341
- Hemorrhoidal tumor**, effect of, on sensation, 36, 37
- Hemiplegia**, 28, 213  
associated movements in, 215  
onset of, 215  
circulatory disturbances in, 215  
complications of, 215  
contraindications in, 215  
from cerebral hemorrhage, 211  
gut in, 234  
hemianesthesia in, 217  
hysterical, 637  
in brain tumor, 265  
in cerebral softening, 225  
in cerebral pulse of children, 218  
position of upper extremity in, 215
- Hemiplegic gut**, 234
- Hemiplegic state**, 233. *See also Hemiplegia*
- Hemorrhage, cerebellar**, 218  
cerebral, 207  
apoplectic state in, 209  
clinical forms of, 217  
course of, 216  
diagnosis of, 218  
differential diagnosis of, 228  
etiology of, 209  
hemiplegia from, 215  
pathological anatomy of, 208  
prognosis of, 219  
sensory disturbances in, 212  
symptoms of, 219  
treatment of, 220  
triglar disturbances in, 212
- Extradural spinal**, 282  
extraped, 76  
into spinal cord, 362. *See also Hemorrhage*  
into tumor of brain, 265  
intraped, 77  
meningeal, 76  
pituitary, 74  
spinal meningial, 282  
diagnosis of, 283  
etiology of, 282  
anatomical anatomy of, 283  
prognosis of, 284  
symptoms of, 283  
treatment of, 284  
subdural spinal, 282
- Hemorrhagic cerebellar ataxia**, 679
- Cerebrospinal syphilis**, 301  
degeneracy, 699  
spastic paraplegia, 476

- Hereditary spastic paraplegia, progressive**  
*of*, 477  
 symptoms of, 476  
 treatment of, 476  
 (transverse, chronic), 369
- Herdity in etiology of general paresis**, 823
- of insanity**, 397
- neuronic importance of**, 18
- Herpes labialis in leptomeningitis**, 83
- nasal**, 339  
 hyperaemia in peripheral nerves in, 337  
 spinal cord in, 337  
 diagnosis of, 338  
 distribution of, 338  
 etiology of, 336  
 pathology of, 337  
 symptoms of, 337  
 treatment of, 339
- Herpeticum simplex**, 339
- Heterophrenia**, 63
- Hiccup**, 145
- Hile-bornal disease**, 339
- Hip-roofed palate**, 709, 712
- Histology, family, in insanity**, 767  
 of patient in insanity, 767
- Horizontal limitation of cord lesions**, 351
- Hot baths in combined sclerosis of cord**, 467  
 in leptomeningitis, 87  
 in multiple neuritis, 335  
 in tubercular leptomeningitis, 95
- Huntington's chorea**, 578  
 disease, 578  
 diagnosis of, 579  
 etiology of, 578  
 morbid anatomy of, 578  
 symptoms of, 579
- Hutchinson's bark**, 715
- Hydrocephalus cys.**, 92
- Hydrocephalus**, 271  
 acute, 90. See also *Leptomeningitis*,  
*tubercular*.  
 course of, 273  
 diagnosis of, 273  
 etiology of, 271  
 external, 271  
 internal, 271  
 morbid anatomy of, 272  
 prognosis of, 274  
 symptoms of, 272  
 treatment of, 274
- Hydratyldehyde**, 387
- Hydrophobia**, 364  
 diagnosis of, 365  
 morbid anatomy of, 364  
 Negri bodies in, 364  
 symptoms of, 364  
 treatment of, 366
- Hydrotherapy in exophthalmic goiter**, 537  
 in insanity, 783  
 in psychasthenia, 613
- Hysteria in epilepsy**, 867  
 in etiology of insanity, 724
- Hysteria in mania**, 738  
 in mental disease, 739  
 in hysterics, 581  
 in Parkinson's disease, 588  
 in senile dementia, 822  
 in vertigo, 150
- Hysterism in epilepsy**, 867  
 in etiology of insanity, 724  
 in mania, 738  
 in mental disease, 739
- Hypersensit.**, 531
- Hypertonia**, 67
- Hypertonus**, 55  
 in mental disease, 744  
 in tabes dorsalis, 441
- Hypertonus-cerebral**, 330. See also *Cerebral hyperaemia*.
- Hypertonus**, 55  
 auditory, 65, 131  
 from lesions of spinal cord, 349  
 hysterical, 627  
 in mental disease, 743  
 in multiple neuritis, 324
- Hyperkinesia**, 744
- Hyperostosis max.**, 555
- Hyperostosis**, 546
- Hyperthyroidism**, 529
- Hypertonus of muscles**, 32
- Hypertrophic neuritis**, 355
- Hypertrophy, localized**, 357  
 sacrospinous, 358
- Hypodermis**, 744
- Hysteresis**, 682  
 in insanity, 739  
 in treatment of hysteria, 646  
 methods of inducing, 682  
 uses of, 683
- Hypnotism, methods of**, 682
- Hypokaliæmia in insanity**, 763
- Hypokaliæmic myelomalacia**, 799  
 paralysis, 763
- Hypokaliæmia**, 754  
 effect of, on actions, 764  
 waste, 821
- Hypoglossal nerve, anatomical conditions of**, 152  
 diseases of, 152  
 paralysis, 153  
 spasms, 152
- Hypoglossus, affections of, in leptomeningitis**, 83
- Hypomnesia**, 736
- Hypophysis cerebri, overactivity of**, 566  
 transneuronic-related to, 566  
 underactivity of, 566
- Hypopneumonia**, 596
- Hypotonus of muscles**, 32
- Hysteria**, 618, 800  
 abortion in, 629  
 accidents in, 629  
 achronatopon in, 623  
 agnaphia in, 640  
 anoxia in, 618, 628  
 ansthenia in, 621  
 distribution of, 624  
 peculiarities of, 625  
 angina pectoris in, 640



**Hysteria, anorexia in,** 641

- anuria in, 641
- aplasia in, 640
- are *de corde* in, 642, 653
- astasia abasia in, 638
- attacks of ecstasy in, 635
  - of sleep in, 635
- aura of, 639
- epistalsia in, 639
- clonic phase of, 639
- contractures of, 639
- cough in, 638, 640
- course of, 642
- cuvage in, 638
- dermatographism in, 643
- diagnosis of, 644
- digestive apparatus in, 641
- dischromatopsia in, 622
- drophagia in, 641
- dyssmia in, 641
- epileptical attacks in, 635
  - period of, 639
- etiology of, 619
- fever of, 642
- glitters in, 639, 635
- grand attacks of, 639
- heming in, 622
- hemiplegia in, 647
- hyperosthosis in, 627
- hypochondriacity in, 628
- in etiology of mania, 727
- isolated attacks of, 634
- isomorphia in, 637
- motor accidents of, 636
- movements in, 627
- mucous atrophy in, 644
- nocturnal spasm in, 635
- paralysis of, 636
- period of ebullition, 633
  - of delirium, 634
  - of personal attitudes, 631, 634
- phase of convulsions of, 633
  - of great movements of, 633
  - of resolution of, 631, 632
- prognosis of, 641
- phos in, 638
- pulmonary congestion in, 641
- reflex in, 625, 626, 639
- respiratory affection in, 640
- rhythmical spasm in, 635
- salivary chaos in, 638
- sensory oscillations of, 629
- stimulation in, 628
- stool in, 622
- symptomatic attacks in, 635
- spontaneous jerks in, 627
- special senses in, 622
- spinal irritability in, 640
- stigmas of, 621
  - mental, 628
  - motor, 627
  - sensory, 621
- sympoms of, 620
- sympot attacks of, 635
- taste in, 622
- tonic attacks in, 635
- tics in, 638

**Hysteria, tonic phase of,** 639

- torticollis in, 635
  - trance in, 635
  - treatment of, 643
    - general, 645
    - special, 646
  - tremors in, 639
  - trophic accidents of, 642
  - lymphatics in, 641
  - urinary apparatus in, 641
  - vascular accidents of, 642
    - change in, 629
  - vertiginous attacks in, 635
  - visceral accidents of, 640
  - tetralgia in, 640
  - vision in, 622
- Hysterical anesthesia,** 57, 621
- distribution of, 621
  - possibilities of, 625
  - remot, 645
  - contractures, 626
  - fever, 642
  - hyperesthesia, 627
  - manity, 660
  - paralysis, 701
  - pseudomeningitis, 638
  - tics, 634
  - tremors, 629
- Hysterogenic point or zone,** 627
- Hysterosarcoloma,** 647, 666
- Idea in myelitis,** 572
- Idling in leptomeningitis,** 87
- Idling in ventral hemorrhage,** 284
- Idling in spinal leptomeningitis,** 281
- Idea-association, actions induced by dis-**
- orders of, 749
  - disorders of, 748
- Ideas, accented flow of,** 751
- defective evolution of, 745
  - disrupted flow of, 752
  - disorders of, 744
  - impulsive, 750
- Ideas, associative faculty,** 253, 368, 375
- attention in, 381
  - activity and passivity in, 385
  - classification of, 368
  - clothing in, 387
  - consciousness and personality in, 391
  - defined, 368
  - destructiveness in, 385
  - diagnosis of, 382
  - direction of attention in, 310
    - of eye in, 311
    - of hands in, 312
    - of hearing in, 311
    - in sense of touch in, 310
    - of taste and smell in, 341
  - food in, 317
  - general etiology of, 378
  - pathological anatomy of, 394
  - sympnology of, 375
  - treatment of, 391
  - hearing in, 376
  - hydrotherapy in, 316
  - instincts in, 383

- Ideacy, intelligence in, 800  
   language in, 888  
   moral and industrial training in, 911  
   socialization in, 917  
   social treatment of, 916  
   Hemiparesis, 875  
   social training in, 915  
   social movements in, 829  
   muscular sensibility in, 879  
   organic sensations in, 881  
   personality in, 890  
   physical culture in, 891  
   physiology in, 887  
   play in, 885  
   preconception in, 882  
   progress of, 886  
   psychological evolution in, 894  
   reflection in, 883  
   responsibility in, 893, 894  
   right- and left-handedness in, 890  
   sentiments in, 884  
   sight in, 878  
   smell in, 879  
   special aptitudes in, 884  
   surgical treatment of, 916  
   tactile pain in, 879  
   taste in, 879  
   teaching of blindness in, 912  
    language in, 913  
    to walk in, 912  
   thermic sensibility in, 879  
   tinnitus, 875  
   voluntary movements in, 881  
   will in, 893  
 Idiopathic muscular atrophy, 38  
   progressive muscular atrophy, 415.  
   *See also* Progressive muscular atrophy.  
 Idols, education of, 909  
   sacred, 875  
 Illegitimacy, 311  
 Illness, the examination of, 29  
 Illness, 742  
   origin of, 742  
 Imagination in etiology of insanity, 720  
 Imperative ideas, 736  
 Impressibility in hysteria, 629  
 Insanities delirium, 726  
 Insanities, 725  
 Incontinence, urinary, 25  
 Incapacitation, detection of, 31  
 Incubus, 679  
 Indifference in insanity, 761  
 Indifferent electrode, 42  
 Indiscriminate card lesions, 392  
 Infantile cerebral palsy, 295. *See also*  
   Cerebral palsy of children.  
   paralysis, 388. *See also* Poliomyelitis.  
   type of muscular atrophy, 424  
 Infantile, 325  
 Infective neurones, 361  
 Infective virus thrombosis, 234. *See also*  
   *Sinus thrombosis*.  
 Inflammation of brain, 228  
   of nerves, 287. *See* *Neuritis*.  
 Inflammatory virus thrombosis, 234  
 Incontinent apoplexy, 217  
 Inherited syphilis as a predisposing cause  
   to nervous disease, 18  
 Insane, asylums for, 776  
   care, 762  
   isolation of, 779  
   social treatment of, 787  
 Insanity, 687  
   accompanying physical diseases of, 769  
   acute, treatment of, 783  
   age in, 697  
   alcohol in etiology of, 722  
   asthenia in, 768  
   behavior in, 768  
   chronic delirium, 896. *See also*  
   *Paranoia*.  
   circular, 891. *See also* Circular in-  
   sanity.  
   classification of, 690  
   definition of, 687  
   diet in, 785  
   disorders of sensation in, 738  
   drugs in, 790  
   electrotherapy in, 785  
   epileptic, 891  
    acute transitory, 891  
    chronic, 891  
   treatment of, 891  
   etiology of, arteriosclerosis arteries in,  
   726  
    absorption in, 724  
    catarrhs in, 726  
    cysts in, 724  
   epilepsy in, 727  
   exhaustion in, 730  
   gastro-intestinal disorders in, 729  
   genital disease in, 726  
   hysteria in, 724  
   head injuries in, 728  
   heart disease in, 728  
   hysteria in, 727  
   infection in, 725  
   infection in, 725  
   metabolic disease in, 725  
   metopism in, 722  
   metabolic pressure in, 725  
   neuritis in, 721  
   syphilis in, 726  
   organic nervous disease in, 727  
   physiological factors in, 729  
   poverty in, 730  
   postural state in, 721  
   syphilis in, 722  
   syphilis in, 722  
   tuberculosis in, 726  
   various points in, 725  
   examination in, methods, 765, 770  
   general etiology of, 687  
   symptomatology of, 757  
   treatment of, 776  
 Ischemic, 731  
 Heredity in, 687  
 Hydrotherapy in, 781  
 Hypoekthymia in, 763  
 Hysteria, 899  
 manic-depressive, 790. *See also* *Manic-  
   depressive insanity*.  
 manic-depressive in, 789  
 images in, 783



- Insanity, senectutian is, 764  
   moral causes of, 772  
   motor disorders is, 761  
   paralysis is, 761  
   physiology is, 768  
   progressive systematized, 826  
   propylaxis of, 778  
   psychotherapy is, 780  
   reciprocal, 755  
   reflex disorders of, 762  
   ret-ent is, 762  
   secretory disorders is, 762  
   sensory disorders is, 761  
   sex is, 697  
   strain is, 723  
   temperature changes is, 764  
   toxic influences is, 772  
   traumatic, 728  
   trophic disorders is, 762  
   urine is, 763  
   vascular disorders is, 763  
 Insomnia, 676  
   etiology of, 676  
   symptoms of, 676  
   treatment of, 677  
 Insular sclerosis, 181. *See also Multiple cerebropetal sclerosis.*  
 Integument, examination of, 25  
 Intelligence, testing of, 772  
 Intention tremor, 20  
   in multiple sclerosis, 185  
 Intermittent hiccup, 590  
   treatment of, 551  
   urine, 796  
 Intermittent Hiccup, 590  
 Internal capsule, function of, 184  
   lesions of, effect of, on sensation, 57  
   motor paths in, 184  
   sensory paths in, 184  
 Intrajugal hemorrhage, 77  
 Intrapsychic ataxia, 535  
 Invasus symptoms, 192  
 Ixodidion melanopholia, 808  
   course of, 810  
   prognosis of, 810  
   symptoms of, 809  
   treatment of, 810  
 Iodid of potassium in bronchial asthma, 144  
 Iodids in cerebral hemorrhage, 221  
   in cerebrospinal syphilis, 502  
   in general paresis, 833  
   in intermittent hiccup, 551  
   in leptomenigitis, 85  
   in neuritis, 201  
 Iodism, injection of, within dura in tubercular leptomenigitis, 96  
   antidote for leptomenigitis, 85  
 Iridoplegia, 63  
 Iron in clove, 577  
   in multiple neuritis, 335  
   in neurasthenia, 669  
 Irradiation, 743  
 Ironing, 687  
 Iridoplegia, 748  
 Irritative brain lesions, 192  
 Ironing, 687  
 Isolation of insane, 779  
  
 Jacksonian convulsions, 32  
   in cerebral softening, 225  
   for, 384  
 Jace-jack, production of, 34  
 Jendrassik's method of examining knee-jerk, 35  
 Joints, Charcot, 49, 452  
   mobility of, in infantile cerebral palsy, 290  
   testing sensibility of, 54  
   trophic disorders of, 93  
 Judgment, weakness of, 757  
 Jimpers, 616  
 Juvenile atrophy, 424  
   tabes, 458  
   variety of atrophic muscular atrophy, 424  
  
 Karyomeres, 266  
 Kakei, 346, 350  
 Kidney-shaped skull, 704  
 Kernig's sign in leptomenigitis, 82  
   in urticaria, 561  
 Knee-jerk, 36  
   reinforced by Jendrassik's method, 36  
 Kocher's ventral operation in atony, 916  
 Koch's bacillus in leptomenigitis, 79  
 Kosakoff's psychosis, 721  
   urine, 821  
  
 Lability of phenomena, 749  
 Labioglossodaryngeal paralysis, 357. *See also Palaeocerebral inferior sclerosis.*  
 Labyrinth tests, 126  
 Laberithase disturbance in tabes, 400  
 Lachrymal reflex in hysteria, 624  
 Laffing, 68  
 Landolt's rule in testing diplopia, 119  
 Landouze-Bergius type of muscular atrophy, 424  
 Landry's paralysis, 376  
   course of, 379  
   diagnosis of, 379  
   etiology of, 377  
   method anatomy of, 378  
   prognosis of, 379  
   symptoms of, 378  
   treatment of, 380  
 Laryngeal crisis, 119  
 Epilepsy, 142  
   muscle, action of, 140  
   nerves, 129  
   palsies in tabes, 449  
   paralysis, 140  
   abductor, 140  
   adductor, 140  
   complete bilateral, 132  
   diagnosis of, 140  
   of tumors, 141  
   treatment of, 142  
 Epasm, 142

- Laryngeal stroke, 349  
 Larynx, anesthesia of, 342  
 Latah, 616  
 Latent lesions of cerebral cortex, 171  
 Lateropulsion, 563  
 Lathyrism, 477  
 Law of regression, 745  
 Lead in etiology of insanity, 725  
 Lead-cap headache, 662  
 Lead-palsy, 328  
     progress of, 333  
     treatment of, 334  
 Leg sign in tetany, 543  
 Length-breadth index, 704, 707  
 Lenticular degeneration, progressive, 387  
 Lenticular zones, 535  
 Leprosy, Antman, 477  
 Leprosy neuritis, 331  
 Leptoccephalus, 703  
 Leptomenigitis, 78  
     acute, spinal, 278  
     course of, 280  
     diagnosis of, 280  
     etiology of, 278  
     anatomical anatomy of, 279  
     Osler's classification of, 79  
     progress of, 281  
     symptoms of, 279  
     treatment of, 281  
     chronic, 97  
     spinal, 281  
     anatomical anatomy of, 282  
     progress of, 282  
     symptoms of, 282  
     treatment of, 282  
     course of, 84  
     diagnosis of, 85  
     diphtheric variety, 86  
     etiology of, 78  
     headache in, 81  
     Kernig's sign in, 82  
     lesion of pons in, 88  
     pathological anatomy of, 80  
     progress of, 86  
     symptoms of, 81  
     treatment of, 87  
     tubercular, 96  
     course of, 94  
     diagnosis of, 94  
     etiology of, 90  
     headache in, 92  
     pathological anatomy of, 90  
     progress of, 95  
     rhin in, 93  
     symptoms of, 92  
     treatment of, 95  
     varieties of, 80  
 Lesage's sign in sciatic neuritis, 311  
 Lesions of one-half of cord, effect of, 36  
     of spinal cord, anesthesia in, 35  
     combined, in aneurism and ma-  
     chiasma, 468  
 Leschlagie cerebellitis, 341  
 Leukostellia posterior, 429. See also  
     Tuber dorsalis.  
 Life, estimation of, 62  
 Ligaments, trophic disorders of, 40  
 Lightning points in tuber dorsalis, 439  
 Limbs, anomalies of, 719  
 Limp-chorea, 573  
 Limping, intermittent, 550  
     treatment of, 551  
 Lingual spasm, 352  
 Lipoma of brain, 260  
 Lipomatosis, symmetrical, 553  
 Lips, anomalies of, 713  
 Litigation psychosis, 663  
 Little's disease, 252  
 Local aphasia, 547  
     conduction, 55  
     death, 547  
     synopsis, 547  
 Localization, cerebral, 165  
     general considerations of, 165  
     in cerebral cortex, 165  
     motor cortical, 167  
     of cord lesions, horizontal, 351  
     vertical, 347  
     of lesions of cauda equina, 360  
     sensory cortical, 170  
     spinal, 340  
 Localized hypertrophy, 557  
     symptoms, 194  
 Lockjaw, 364  
 Locomotor ataxia, progressive, 429. See  
     also Tuber dorsalis.  
 Leprosy, 708  
 Lumbal points, 311  
     puncture, 27  
     in leptomenigitis, 88  
     in serous meningitis, 97  
     in spinal leptomenigitis, 281  
     in tubercular meningitis, 90  
 Lumbal in epilepsy, 663  
 Lyons, 564  
 Lymphoblast, 565  
  
 MACROCEPHALUS, 702  
 Macrocephalism, 515  
 Mairgksson, 715  
 Magnesium sulphate in chorea, 577  
     in tetanus, 565  
 Make of current, 42  
 Maladie des tics, 614, 757  
 Mandibular muscles, paralysis of, from  
     disease of trifacial nerve, 119  
     reflex, 34  
 Mania, 795  
     acute delirious, 796  
     course of, 797  
     diagnosis of, 797  
     dystimular in, 794  
     etiology of, 792  
     hearing in, 152  
     intermittent, 796  
     mania, 796  
     periodic, 796  
     progress of, 795  
     reasoning, 796  
     recurrent, 796  
     senile, 821  
     transitory, 796  
     treatment of, 797



- Mania, unproductive, 791  
     varieties of, 796  
 Manic stupor, 791  
 Manic-depressive insanity, 799  
     circular type, 803. *See also Circular insanity.*  
     classification of, 792  
     depressive phase, 798. *See also Melancholia.*  
     etiology of, 792  
     manic phase, 791. *See also Mania.*  
     prognosis of, 795  
 Marantic acute thrombosis, 255. *See also Sympa thrombosis.*  
 Massage in anterior poliomyelitis, 326  
     in combined sclerosis of cord, 367  
     in ophthalmic gitter, 357  
     in facial paralysis, 129  
     in infantile cerebral palsy, 255  
     in insanity, 783  
     in interstictal larynx, 351  
     in Landry's paralysis, 389  
     in multiple neuritis, 315, 330  
     in myelitis, 372  
     in neuritis, 291  
     of brachial plexus, 336  
     in psychasthenia, 613  
     in sciatic neuritis, 314  
     in spinal muscular progressive atrophy, 415  
     in tabes dorsalis, 461  
 Massive type of arteriosclerosis, 513  
 Maudsley paralysis, 118  
     spasm, 118  
 Mastoid disease, importance of, 56  
 Masturbation in insanity, treatment of, 799  
 Mathias's hand-dynamometer, 28  
 Maximal points of pain, 28  
 Median nerve, lesions of, 299  
 Medulla oblongata, symptoms of lesions of, 189  
 Megalocephalie, 555  
 Megalocortyly, 719  
 Megalopsia, 719  
 Melancholia, 795  
     agitata, 792, 809  
     anxia, 809  
     attonita, 792  
     course of, 801  
     diagnosis of, 801  
     hypochondriacal, 799  
     irritation, 808. *See also Jacobus melancholia.*  
     parva, 792  
     prognosis of, 799  
     seile, 821  
     simplex, 809  
     stuprosa, 804  
     treatment of, 802  
     varieties of, 801  
 Memory defects, actions induced by, 758  
     disorders of, 759  
     importance of, in diagnosis, 25  
     testing of, 771  
 Memory-pictures, disorders of, 744, 745  
 Memory-trace, 751  
 Ménière's disease, 134. *See also Aural vertigo.*  
 Meningeal hemorrhage, 78  
     spinal, 282  
 Meninges, cerebral, 72  
     spinal, tumors of, 382  
 Meningitis, acute cerebral, 78. *See also Leptomenigitis and Pachymenigitis.*  
     arosa, 96  
     basilar, 91. *See also Leptomenigitis, tubercular.*  
     cerebral, syphilitic, 492  
     symptoms of, 497  
     conspinal, 78. *See also Leptomenigitis.*  
     chronic infantile, 90  
     diphtheric variety, 89  
     course of, 89  
     diagnosis of, 89  
     etiology of, 89  
     prognosis of, 89  
     symptoms of, 89  
     treatment of, 89  
     externa arosa, 96  
     interna arosa, 96  
     pueril, 78. *See also Leptomenigitis.*  
     arosa spinalis circumscripta, 284  
     arosa, 96  
     course of, 97  
     diagnosis of, 97  
     etiology of, 96  
     pathology of, 96  
     prognosis, 97  
     symptoms of, 96  
     treatment of, 97  
     spinal, 279  
     syphilitic spinal, 199  
 Meningocele, 387  
 Meningoencephalitis, spinal, 276  
     syphilitic, 496  
 Meningoencephaloma, 387  
 Meningovascular lesions in nervous system, 191  
 Menopause in etiology of insanity, 752  
 Menstruation in insanity, 764  
 Mental condition of patient, examination of, 22  
     dangers, 687  
     disturbances as a cerebral symptom, 194  
     strain, 721  
     symptoms in tumor of brain, 263  
     torticollis, 150, 434  
 Mergula parasthetica, 367  
 Menstrual irregularities in insanity, 291  
 Memory in cerebrospinal syphilis, 562  
     in etiology of insanity, 725  
     in general paresis, 515  
     in hydrocephalus, 274  
     in leptomenigitis, 88  
     in tabes dorsalis, 460  
 Meridians, 711, 771  
 Merysion, 149  
 Mesencephalic head, 192  
 Metabolic poisons, insanity and, 725  
 Metatarsalgia, 399

- Microcephalus, 761  
 Microcrania, 754  
 Middle-ear disease, 66  
 Migraine, 661  
   cause of, 664  
   diagnosis of, 664  
   etiology of, 661  
   features in, 663  
   ophthalmologic, 662  
   paralysis, 667  
   pathology of, 664  
   prognosis of, 665  
   symptoms of, 662  
   treatment of, 665  
 Minor spasm, 414  
 Miss-hemifield, 177  
   lesion causing, 171  
 Mirror-speech, 70  
 Mirror-writing, 70  
 Misophobia, 611  
 Modal change, 46  
 Megalophakia, 565  
 Mesodial lobe, 825  
 Mesocortical lobe, 63  
 Mesencephalon, 836  
 Mesoplegia, 29  
   in tract of brain, 264  
 Meiosis, 746  
 Mental values of insanity, 732  
   treatment of insane, 787  
 Meibomian tube, 647  
 Meerkat, 715, 736  
 Morphine in exanthematous disease, 382  
   in chorea, 377  
   in etiology of insanity, 725  
   in leptomeningitis, 87  
   in mental disease, 786  
   in migraine, 665  
   in neuralgia, 675  
   in Raynaud's disease, 549  
   in scaly neuritis, 315  
   in tabes dorsalis, 463  
   in tetanus, 365  
 Morphological stigmata of degeneracy, 21  
 Morvan's disease, 389  
 Morvan's disease, 485  
 Motility, 28  
   increased, 30  
   reduced, 28  
 Motor aphasia, 178  
   area of brain, localization of, by electricity, 49  
   disorders in insanity, 761  
   function, anomalies of, 720  
   neurones, 375  
   points of face and neck, 42  
     of lower extremity, 45-47  
     of upper extremity, 43, 44  
   time, 611  
   tracts of brain, 154-191  
 Movements, hallucinations of, 739  
 Movements, associated, 32  
   postplegic, 31  
 Multiple abscesses of brain, 242  
   cerebrospinal sclerosis, 481  
     abdominal reflex in, 485, 489  
     bulbar symptoms of, 487  
 Multiple cerebrospinal sclerosis, cerebral  
   symptoms of, 487  
     course of, 488  
     diagnosis of, 488  
     etiology of, 481  
     forms of, 488  
     intention tremor in, 485  
     morbid anatomy of, 487  
     motor features of, 484  
     prognosis of, 489  
     reflexes in, 485  
     sensory symptoms of, 486  
     symptoms of, 484  
     treatment of, 489  
     trophic disturbances in, 487  
     vertigo in, 487  
     visceral disturbances in, 487  
     visual disturbances in, 487  
   neuritis, 319. See also Neuritis.  
   recurrent, 333  
   remittent, 292  
   paralysis of cranial nerves, 154  
 Muscles, electrical testing of, 41  
   testing of power of, 28, 29  
   trophic disorders of, 40  
 Muscular sense, testing of, 54  
   spasm, idiopathic, 28  
   system, examination of, 28  
 Muscular nerve, lesions of, 267  
 Mutation, 770  
   in dementia praecox, 815  
   in hysteria, 68  
 Myasthenia gravis, 162  
 Myasthenic reaction, 49  
 Myelinic retrovirus, 262  
 Myelitis, 365  
   acute bulbar, 162  
   epidemic, 500  
   atrophy in, 369  
   central, 366  
   course of, 370  
   diagnosis of, 370  
     differential, 371  
   dissiminated, 366  
   etiology of, 366  
   morbid anatomy of, 369  
   motor symptoms of, 368  
   paraplegia in, 373  
   prognosis of, 371  
   reflexes in, 368  
   sensory symptoms of, 368  
   symptoms of, 367  
   transverse, 366  
   treatment of, 372  
   trophic changes in, 369  
 Myoklonia, 387  
 Myoklonus, 379  
   diagnosis of, 380  
   etiology of, 380  
   prognosis of, 381  
   symptoms of, 381  
   treatment of, 381  
 Myoklonus, 379  
   diagnosis of, 337  
   epilepsy, 656  
   prognosis of, 329  
   treatment of, 329





- Neuritis, multiple, electrical changes in, 329  
 etiology of, 326  
 general conditions in, 325  
 lesions in muscles in, 319  
 in nerves in, 317  
 of brain in, 318  
 of spinal cord in, 318  
 involved anatomy of, 317  
 motions of eyes in, 324  
 nutrition in, 325  
 prognosis of, 323  
 pupils in, 324  
 treatment, 331  
 reflexes in, 322  
 special forms of, 327  
 symptoms of, 319  
 mental, 325  
 motor, 318  
 muscular, 319  
 ocular, 324  
 sensory, 321  
 treatment of, 334  
 trophic conditions in, 325  
 vision in, 324  
 of brachial plexus, 304  
 causes of, 304  
 diagnosis of, 305  
 prognosis of, 306  
 symptoms of, 304  
 treatment of, 306  
 of branches of trilateral, 120  
 optic, 105  
 as a symptom, 105  
 parasympathetic, 288  
 prognosis of, 290  
 sciatic, 209  
 causes of, 206  
 diagnosis of, 212  
 double, 212  
 involved anatomy of, 310  
 symptoms of, 210  
 tender points in, 311  
 treatment of, 314  
 symptoms of, 208  
 syphilitic, 301  
 treatment of, 290  
 Neuritis of brain, 260  
 Neuromata, 292  
 amputation, 292  
 avulsion, 292  
 cellular, 292  
 diagnosis of, 293  
 delirium, 292  
 etiology of, 293  
 fibrillar, 292  
 ganglion, 292  
 multiple, 292  
 myeloid, 292  
 prognosis of, 291  
 symptoms of, 293  
 traumatic, 292  
 treatment of, 294  
 Neuropsychoses, 569  
 Neuroretinitis, 105  
 Neuroses, 505  
 classification of, 505  
 Neuroses, fatigue, 503  
 following trauma, 500  
 glandular, 506  
 hypertrophic, 555  
 infectious, 561  
 motor, 558  
 occupational, 50, 503, 509  
 vascular, 547  
 Neurotubes, 459  
 Neuritic bendure in diagnosis, 15  
 Nightmare, 679  
 Night-terrors, 25, 679  
 Ninth nerve. See *Glossopharyngeal nerve*.  
 Nitrate of strychnin in spinal progressive muscular atrophy, 115  
 Nitrite of amyl in bronchial asthma, 145  
 in tetanus, 393  
 Nitroglycerin in migraine, 665  
 Nocturnal enuresis, 679  
 epilepsy, 653  
 Nodding system in hysteria, 658  
 Nosis, 241  
 Normal child, development of the faculties of, 903  
 Nose, anomalies of, 714  
 Nosebleed after middle Me, 250  
 Nucleate of soda in general paresis, 832  
 Nutrition, abnormal variations in, 39  
 Nyctagnus, 111  
 Olfactory lobe, 173  
 Olfactory nerve, lesions of, 307  
 Occupational neuritis, 50, 503, 509  
 predisposing to nervous disease, 20  
 Ocular muscle, spasm of, 114  
 muscles, action of, 108  
 nerves, anatomical considerations of, 107  
 diseases of, 107  
 palsies, 109  
 causes of, 111  
 diagnosis of, 110  
 location of lesion in, 112  
 treatment of, 115  
 Oculomotor apparatus, double corneal representation of, 108  
 Olfactory nerve, diseases of, 50  
 Oligodactyly, 718  
 Oligoscleritis, 719  
 Oliguria in insanity, 705  
 Operative psychosis, 323  
 Ophthalmic test in tubercular keratitis, 95  
 Ophthalmoplegia externa, 63  
 interna, 63  
 progressive, 155. See also *Poliomyelitis superior chronic*.  
 Ophthalmoplegia migrans, 662  
 Ophthalmoscope, importance of, to neurologist, 63  
 Ophthalmoscopic picture in anastrotic family disease, 253  
 Orthognathism, 705  
 Ophthalmoscopy in cerebellar disease, 190  
 Optum in epilepsy, 661



- Optic in insanity, 796  
 in mania, 798  
 in melancholia, 901  
 in Parkinson's disease, 585  
 in torticollis, 150
- Opiate-bromid treatment in epilepsy, 866
- Optic atrophy, 263  
 nerves: anatomy of, 100  
 atrophy of, 40, 196  
 diseases of, 99  
 lesions of, 100, 101  
 neuritis as a syngonon, 195  
 in tumors of brain, 265  
 thalamic, function of, 187  
 lesions of, 104, 187  
 tract, lesions of, 100-101
- Orbits: asymmetry of, 708
- Orientation, patient's, 771
- Orthognathism, 795
- Oster's classification of acute leptomen-  
 ingitis, 79
- Osteo-arthropathia hypertrophicans  
 (osteosarcoma), 536
- Osteo-arthropathy, hypertrophic, 536
- Otentat of brain, 260
- Otitis externa, 762
- Oxaluria in insanity, 763
- Oxycephalia, 703
- P**
- Pachymeningitis, 74
- Pachymeningitis, 74  
 cervical hypertrophica, 277  
 externa, 74  
 spinalis, 276  
 haemorrhagica, 74  
 interna, 74  
 course of, 75  
 diagnosis of, 76  
 etiology of, 74  
 pathological anatomy of, 74  
 spinalis, 277  
 symptoms of, 75  
 treatment of, 76
- Pain, areas of, cerebral disease and, 58  
 as a cerebral symptom, 195, 195  
 as a symptom, 57  
 description of, by patients, 62  
 in agria pectoris, 147  
 in brain disease, 62  
 in herpes zoster, 338  
 in inflammation of suboccipital nerves,  
 305  
 in lesions of ocula optica, 300  
 in myelitis, 367  
 in neuritis, 289  
 in neuralgic neuritis, 310  
 in spinal tumors, 284  
 in syphilitic meningitis, 460  
 in tabes dorsalis, 429, 440  
 maximal points of, 58  
 sense: testing of, 51
- Painful stretching symptom in lesions of  
 cranial system, 360
- Palate, mesoventral, 700, 712  
 left, 710  
 deformities of, 708
- Palate, down-shaped, 700, 711  
 flat-roofed, 700, 711  
 Gothic, 700, 710  
 lip-roofed, 700, 712  
 with horseshoe arch, 700, 710
- Palpebral reflex, 32
- Palpitation, cardiac, 146  
 in brain tumor, 190
- Palmis, contracted, of nerves of arm, 202  
 in tabes dorsalis, 428  
 sleep, 181
- Palsy, Bell's, 123  
 drier's, 380. See also *Criens stis-*  
*mo*  
*activans*, 393  
 wasting, 408
- Paracanthoma in leptomeningitis, 83
- Papilla, optic, diseases of, 104
- Papillitis, 104  
 in tumor of brain, 265
- Papillomectomy, 365
- Paradoxical constriction, 37
- Paralis in facial hemiatrophy, 342
- Paraglossa, 67
- Paraplegia, 69
- Paralyzed in mania, 798  
 in mental disease, 787  
 in senile dementia, 822
- Paralysis, acute, ascending, 370. See  
 also *Landy's paralysis*  
 agitante, 582. See also *Parkinson's*  
*disease*  
 Brown-Séquard, cord lesion in, 60, 67  
 luffus, anthesis, 642  
 cerebral, of children, 246  
 chronic nuclear oculis, 453. See also  
*Polioencephalitis superior chronic*  
*syphilitic*, 329  
 epidemic, 388  
 focal, 123. See also *Facial paralysis*  
*familial periodic*, 501  
 from brain disease, 244  
 from cerebral haemorrhage, 226  
 from cord lesion, 347  
 hypoglossal, 153  
 in anterior poliomyelitis, 205  
 in cerebral softening, 225, 226  
 in hysteria, 636  
 in insanity, 763  
 in multiple neuritis, 319  
 in myelitis, 308  
 in syphilitic meningitis, 460  
 in tabes dorsalis, 428  
 infantile, 388. See also *Poliomyelitis*  
*labio-glossolaryngeal*, 157. See also  
*Polioencephalitis chronic*  
*Landy's*, 376  
 laryngeal, 140. See also *Laryngeal*  
*paralysis*  
 maxillary, 118  
 multiple, of cranial nerves, 154  
 ocular, 100. See also *Ocular palsy*  
 of anterior cranial nerve, 207  
 of auditory nerve, 132  
 of circumflex nerve, 287  
 of facial nerve, 121  
 of median nerve, 300

- Paralysis of transverse spinal nerve, 288  
 of olivator nerve, 367  
 of pharic nerve, 245  
 of posterior thoracic nerve, 296  
 of sciatic nerve, 298  
 of spinal accessory nerve, 151  
 of superior gluteal nerve, 307  
 of suprascapular nerve, 297  
 of tongue, 453  
 pharyngeal, 428  
 progressive bulbar, 157. See also  
*Poliomyelitis inferior chronic*.  
 pseudobulbar, 162  
 pseudohypertrophic, 424  
 syphilitic spinal, 300  
 ulnar, 300
- Parsylvic rhomb, 575  
 dementia, 480, 819, 822  
 definition of, 822  
 diagnosis of, 831  
 differentiation of, from alcoholism,  
 831  
 from cerebrospinal syphilis, 832  
 from multiple sclerosis, 831  
 from neurotoxicosis, 831  
 duration and prognosis of, 830  
 etiology of, 825  
 mental symptoms of, 825  
 pathological anatomy of, 833  
 physical symptoms of, 825  
 prodromal period of, 824  
 symptomatology of, 824  
 terminal period of, 830  
 treatment of, 834  
 trigeminal, 367
- Parasomnia, 69, 177
- Parasymphysis, 579
- Parasymphysis, multiple, 580
- Parasymphysis, congenital, 580
- Parasomnia, 830  
 alcoholic, 725  
 course and prognosis of, 830  
 definition of, 830  
 nature, 830  
 etiology of, 837  
 expansive period of, 840  
 hallucinations, 836  
 chronic, 830  
 monitory, 840  
 neural anatomy of, 839  
 period, 710  
 premonitory period of, 838  
 prodromal period of, 837  
 querulous, 840  
 reformations, 840  
 religious, 840  
 simplex, 836  
 chronic, 830  
 symptomatology of, 837  
 treatment of, 839  
 varieties of, 836
- Parasymphysis, dementia, 818
- Parasymphysis, 176, 179
- Parasymphysis, 69
- Paraplegia, 29, 374. See also Paraplegia  
 state.  
 static, 464
- Paraplegia, static, syphilitic, 500  
 from hematomyelia, 364  
 hereditary spastic, 376. See also  
*Hereditary spastic paraplegia*.  
 in myelitis, 375  
 in spinal tumors, 384
- Paraplegic gait, 374  
 static, attitude in, 375  
 etiology of, 375  
 gait in, 374  
 in myelitis, 375  
 prognosis of, 376  
 reflexes in, 375  
 symptoms of, 375  
 treatment of, 376
- Parasympathetic diseases, 501  
 cerebral, 503  
 hereditary, 503  
 lesions in nervous system, 494  
 nervous disease, 499
- Parathyroid glands, neoplasms pos-  
 sessed by, 518
- Parathyroidism, neuritis, 288
- Paresis, general, in cerebral hemorrhage,  
 219
- Paresis in multiple neuritis, 321  
 in neuritis, 268
- Paresis, 35
- Parkinson's disease, 582, 588  
 cause of, 587  
 diagnosis of, 588  
 etiology of, 582  
 mental state in, 587  
 neural anatomy of, 582  
 muscular rigidity in, 584  
 palsy in, 587  
 sensory disturbances in, 587  
 symptoms of, 583  
 treatment of, 588  
 trembling in, 589
- Past illness, investigation of, 19
- Patellar point, 311  
 index, 31
- Pathological drunkenness, 725
- Patent, antecedents of, 18  
 examination of, 17-71  
 observation of, in insanity, 708
- Pavor nocturnus, 679
- Pellagra, 477  
 treatment of, 480
- Pellucida, 477
- Pernicious in brain disease, 195
- Pernicious, cerebral, 302
- Pernicious, 288
- Periodical mania, 706  
 swelling, 552
- Peripheral nerves, trophic disease of, 49  
 paralysis in insanity, 761
- Peripheral nerve, lesions of, 308  
 variety of idiopathic muscular atrophy,  
 424
- Persecution, delusion of, 754
- Personal history, 11
- Peterson's collars, 705, 706
- Peto mal, 654
- Pharyngeal crisis, 449  
 paralysis, 129



- Pharyngeal reflex, 34  
spasm, 129
- Pneumonia in arachnoiditis, 512  
in syphilis, 689  
in tuberculous, 463
- Pneuroencephalitis in determination of relations of ventricular outlets, 274
- Pneumonia, 719
- Phosphaturia in insanity, 763
- Phosphorus as a symptom, 62  
in leptomeningitis, 82
- Purkinje nerve, lesions of, 285
- Physical examination, 24  
strain, 723
- Physiology of patient, 24
- Physiological factors in etiology of insanity, 729
- Pia mater, 72  
inflammation of, 78. *See also* *Leptomeningitis*  
viral, 72
- Pit hemorrhage, 76  
spasm, 72
- Pituitary in neural vertigo, 128  
in nervous deafness, 155
- Pituitary gland, 517  
tumors of, 518  
syndromes, 517
- Piquet's test in tubercular meningitis, 55
- Pituitary extract in adipose deformity, 517  
feeding in adipose deformity, 517  
gland, 506  
tumors with arachnoiditis, 269  
with hydrocephalus, 271
- Pneuroencephalitis, 701
- Plantar nerve, external, lesions of, 399  
internal, lesions of, 399  
points, 311  
reflex, 37
- Platycarpus, 705
- Platyrrhini, center for, 599
- Pneumococcus in leptomeningitis, 79
- Pneumogaster nerve. *See* *Vagus*
- Polar change, 47
- Poliocencephalitis, modified forms of, 462  
infantile chronic, 457  
course of, 464  
diagnosis of, 461  
etiology of, 457  
neural anatomy of, 458  
symptoms of, 458  
treatment of, 461
- superior, acute, 457  
chronic, 455  
course of, 456  
diagnosis of, 456  
etiology of, 455  
pathological syndrome of, 455  
symptoms of, 455  
treatment of, 456
- Poliocencephalomyelitis, 388
- Poliomyelitis, acute anterior, 388  
ascending form, 396  
course of, 395  
deformity in, 394, 395  
diagnosis of, 396
- Poliomyelitis, acute anterior, etiology of, 399  
course of, 395  
neural anatomy of, 399  
progress of, 396  
symptoms of, 395  
treatment of, 397
- chronic, 406
- Poliomyelitis, 719
- Polyoma, 719
- Poliomyelitis, 516. *See also* *Neuritis*, *multiple*
- Polyoma in insanity, 763
- Pons Varoli, function of, 485  
syndromes of lesions of, 485
- Posterior nerve, external, lesions of, 398  
internal, lesions of, 398  
points, 311
- Posterior column of cord, effect of lesions of, 331  
horn of cord, effect of lesions of, 332  
roots of cord, effect of lesions of, 332  
spinal arteries, 346
- Posterior lateral columns, 404. *See also* *Combined columns of cord*
- Postlagic movements, 31
- Precedal anxiety, 747  
light, 747
- Precedal paroxysmal condition, 822
- Present condition of patient, 24
- Pressure sense, testing of, 51
- Proctitis, spastic, 539
- Prognosis, 705
- Progressive bulbar paralysis, 457. *See also* *Poliocencephalitis superior chronic*  
general paralysis, 822. *See also* *Paralytic diseases*  
bulbar degeneration, 457  
chronic ataxia, 429. *See also* *Tuber sclerosis*
- muscular atrophy, 467  
disability, 415  
course of, 425  
etiology of, 416  
neural anatomy of, 417  
progress of, 426  
symptoms of, 418  
treatment of, 426  
varieties of, 424
- spinal, 407  
course of, 411  
diagnosis of, 411  
differential diagnosis of, 414  
etiology of, 408  
neural anatomy of, 408  
progress of, 415  
symptoms of, 408  
treatment of, 416  
varieties of, 411
- with cord lesions. *See* *Progressive muscular atrophy, spinal*  
without cord lesions. *See* *Progressive muscular atrophy, idiopathic*  
dystrophy, 415
- ophthalmoplegia, 433. *See also* *Poliocencephalitis superior chronic*

- Progressive spastic ataxia, 444. See also  
Combined sclerosis of spinal cord;  
systematized insanity, 838. See also  
Parosmia.
- Projectile vomiting as a symptom, 194
- Propulsion, 565
- Protopathic sensibility, 55
- Potomania, 289
- Pseudoparesis, 753
- Pseudoparalysis, 755
- Pseudotaxic cerebellum, 670
- Pseudotumor parietalis, 162
- Pseudocerebral tumor, 207
- Pseudochorea, 753
- Pseudodementia, 748
- Pseudohyperreflexic paralysis, 424
- Pseudomania parasthetica, 216
- Pseudomeningitis, hysterical, 639
- Pseudoparalysis, congenital atonic, 425
- Pseudoparalysis, 753
- Pseudoparesis, hysterical, 497
- Pseudotumor, 419
- Psychanalysis, 773
- Psychodilemma, 606, 610  
course of, 612  
diagnosis of, 613  
etiology of, 612  
onset of, 612  
progress of, 613  
termination of, 613  
treatment of, 613
- Psychic degeneration of epileptics, 802  
equivalent of epileptic attack, 655
- Psycho-analysis in psychothemia, 612
- Psychogenia, 800
- Psychogeny, 800
- Psychopulver, 687
- Psychosis, 687  
alcoholic, 721  
Korsakoff's, 721  
Brighton, 687  
senile, 499, 822  
Korsakoff's, 821  
traumatic, 667
- Psychotherapy, 780
- Prose as a symptom, 92  
sleep, 682
- Puberty in etiology of insanity, 730
- Puerperal state in etiology of insanity, 731  
tetanic, 562
- Pulmonary branches of vagus, affection of, 143
- Pulse in leptomeningitis, 83  
in poliomyelitis inferior chronica,  
160  
in spinal leptomeningitis, 280  
in tubercular leptomeningitis, 92
- Puncture, spinal, 27
- Pupal irregularities of, 63
- Pupillary reflex, 31
- Pupils in leptomeningitis, 83  
in multiple neuritis, 324  
in tubercular meningitis, 93
- Purulent meningitis, 78. See also *Leyde*  
*meningitis*.
- Pyramidal tracts, effect of lesions of,  
131
- Qualitative change, 47
- Quantitative change, 90
- Quincke's disease, 552  
basilar puncture, 27  
in serous meningitis, 97  
in spinal leptomeningitis, 281
- Quins in aural vertigo, 158  
in chorea, 577  
in multiple neuritis, 335  
schmids, 489  
in neuritis, 672
- Rabies, 564
- Radium in syringomyelia, 406
- Rapies ichthyolepis, 809
- Raynaud's disease, 547  
course and prognosis of, 548  
diagnosis of, 548  
etiology of, 547  
symptoms of, 548  
treatment of, 549
- Reaction, homonymous pupillary, 633  
myotheric, 49  
aptonic, 48  
of degeneration, 30  
in facial paralysis, 126  
partial, 47
- Recessus, 25  
in cerebral syphilis, 497
- Recessing mass, 796
- Reciprocal insanity, 753
- Rectus clemis, 37  
reflex, 37
- Recurrent mass, 796  
multiple neuritis, 331
- Red cerebral softening, 723
- Referred sensation, 54
- Reflected tone, 746
- Reflex, abdominal, 35  
Achilles' tendon, 37  
anal, 38  
sphincteric, 38  
bulbo cavernosus, 448  
ciliary, 33  
cervicogenic, 38  
disorders in insanity, 762  
epigastric, 35  
heel tendon, 37  
mamillary, 34  
palpebral, 33  
patellar, 38  
pharyngeal, 34  
plantar, 37  
pupillary, 33  
rectus, 37  
sphincter, 38  
supraclavicular, 33  
triceps, 35  
vulv, 38, 448
- Reflexes, 32  
in anterior poliomyelitis, 394  
in hysteria, 625, 628, 636  
in lesions of spinal cord, 349  
in multiple cerebrospinal sclerosis, 485  
tetanic, 322  
in myelitis, 368



- Reflexes in syphilitic epilepsy, 656  
 in torpid state, 325  
 in progressive muscular atrophy with  
 cerebral lesions, 418  
 in spinal tumours, 384  
 in tubes dorsalis, 442  
 of lower extremity, 35  
 of upper extremity, 34
- Refraction, errors of, importance of, 63
- Refusal of food, management of, 789
- Regeneration of a divided nerve, 280
- Regression, law of, 745
- Resection of nerves in tubes dorsalis,  
 462
- Residues, importance of, 26
- Respiration, in leptomeningitis, 84  
 in tubercular leptomeningitis, 92
- Respiratory organs, examination of, 24
- Ros is multiple sclerosis, 335  
 in leucemia, 291  
 in acetic neuritis, 314
- Rotatory, 782
- Retina, diseases of, 164
- Retinitis, 104
- Retrigade anaesthesia, 751
- Retropulsion, 365
- Reversion, 698
- Rheumatism, relation of, to chorea, 508
- Right-handedness, cause of, 162
- Rigidity in tubercular leptomeningitis, 92  
 muscular, in leptomeningitis, 82
- Rinne's test, 65
- Robertson's pupillary sign in tubes, 445
- Roegen rays in brain disease, 196
- Romberg, sign of, 439  
 symptoms, 30
- Rudinski's sign in leptomeningitis, 82
- Rumination, 119
- Sacculus, line, 172
- Salivary gland, role in auto-intoxication,  
 785
- Salivary glands in adiposis dolorosa, 517  
 in anterior poliomyelitis, 397  
 in brachial plexus neuritis, 308  
 in facial paralysis, 129  
 in Landry's paralysis, 380
- Saline solution in cerebral softening, 290  
 injection in acute neuritis, 314
- Salk in auto-intoxication, 785  
 in epilepsy, 660, 867  
 in multiple neuritis, 335  
 in myelitis, 372
- Salivary glands in hysteria, 638
- Salvarsan as preventive of paresis, 770  
 in cerebral syphilis, 362  
 in paralytic dementia, 835  
 in tubes dorsalis, 464
- Sarcoma of brain, 257
- Saturnine speech, 68
- Scaphocephalus, 704
- Schizothymia, 817
- Sciatic nerve, great, lesion of, 308  
 lesions, 308. See also Neuritis  
 sciatica, 311
- Sclerodactylic, 539
- Scleroderma, 539  
 etiology of, 539  
 symptoms of, 540  
 treatment of, 540
- Sclerosis altharum, 539
- Sclerosis en plaques, 481
- Scleroses of spinal cord, contained, 661
- Sclerosis, argyrotrophic lateral, 497  
 disseminated, 483  
 multiple cerebrospinal, 481. See also  
 Multiple cerebrospinal sclerosis  
 of posterior columns of cord, 429. See  
 also Tubes dorsalis  
 of spinal cord from vegetable neuro-  
 cists, 477  
 postherpetic, 664. See also Con-  
 fused sclerosis
- Scoliosis, acute, 331
- Scolio disease, 784
- Scoliosis, central, 168
- Scombrura, 64
- Scurvy, policy, 563
- Scurvy, Alpine, 477
- Secondary dementia, 819. See also De-  
 mentia
- Secondary disorders in insanity, 762
- Sedatives in leptomeningitis, 82
- Seile disease, 380  
 dementia, 819, 820. See also De-  
 mentia  
 hypochondriasis, 821  
 Korsakoff's psychosis, 823  
 mania, 823  
 melancholia, 821  
 paranoid condition, 821  
 psychosis, 823, 822
- Serum in etiology of insanity, 762
- Sensation, disorders of, in insanity, 758  
 intensity of, 743  
 qualitative, 738  
 time of, 742  
 general consideration of, 49  
 reduced, 34  
 testing and examination of, 38
- Sensibility, deep, 55  
 specific, 55  
 hallucinations of, 738  
 illusions of, 742  
 prefrontal, 55
- Sensory conditions, 49  
 cerebral localization, 179  
 disorders, actions induced by, 758  
 in insanity, 761  
 disturbances from brain lesions, 193  
 features in insanity, 290  
 function, anomalies of, 751  
 path, 184  
 time, disorders of, 743
- Serological tests for syphilis in paralytic  
 dementia, 835
- Serous meningitis, 96. See also Menin-  
 gitis, serous
- Serum, antineurotic, in chorea, 507
- Flaccid, in diploic variety of  
 meningitis, 89  
 in leptomeningitis, 88
- Seventh cranial nerve. See Facial nerve.

- See in History of insanity, 167  
 Shaking palsy, 347  
 Shell shock, 635, 640, 696  
 Shingles, 236. See also *Herpes zoster*.  
 Shoulder, center for movements of, 169  
 Sight, affection of, in tabularis leptomeningitis, 32  
     extrinsic of, 32  
     inflammation of, 729  
 Sign, Kérat's, in leptomeningitis, 82  
     in tetanus, 341  
     in strychnine, 545  
 Signal symptoms, 72, 181  
 Strabismus in epilepsy, 806  
 Strabismic amblyopia, 212  
     inborn, 214  
     acquired, 215  
     lateral, 216  
     longitudinal, 217  
     symptoms of, 215  
     treatment of, 217  
 Strabismic, 211  
     diagnosis of, 211  
     prognosis of, 214  
     symptoms of, 211  
 Stripes, cerebral, anatomy of, 211  
 Sixth nerve, anatomical considerations of, 108  
     effect of division of, 109  
 Sten, anomalies of, 729  
     trophic disturbances of, 39  
 Sleep, conditions favoring, 675  
     disorders of, 674  
     quiescence, 678  
     importance of, in diagnosis, 23  
     palsy, 298, 684  
     physical lesions of, 674  
     prose, 682  
     requirements for, 675  
     neurotic disorders of, 680  
     nervous disorders of, 678  
 Sleeping sickness, 241, 681  
 Skull, center for, 171  
     examination of, 60  
     inflammation of, 729  
     lesions of, 744  
     use of, 99  
 Sals, maculate of, in paralytic dementia, 835  
 Softening, cerebral, 222. See also *Cerebral softening*.  
 Somnambulism, 678  
 Somnolence, 679  
 Sounds, subjective, 66  
 Space sense, disturbance of, 66  
 Spasmodic asthma, 143. See also *Asthma, bronchial*.  
     tubes, 404  
     torticollis, 149, 340  
     wryneck, 149  
 Spasmodic point or zone, 327  
 Spasms, 31  
     clonic, 31  
     facial, 122, 616  
     from cord lesions, 347  
     hypoglossal, 132  
     hyperal rhythmic, 638  
 Spasms, idiopathic muscular, 38  
     laryngeal, 142  
     lingual, 132  
     masticatory, 118  
     nasal, 614  
     occlusion, 293  
     of ocular muscle, 114  
     of spinal accessory nerve, 149  
     pharyngeal, 139  
     sialic, 31  
 Spastic paraplegia, hereditary, 476. See also *Hereditary spastic paraplegia*.  
 Spinal senses, examination of, 62  
 Speech, anomalies of, 721  
     center for, 170, 174  
     examination of, 67  
 Sphincter reflex, 35  
 Spina bifida, 388  
     diagnosis of, 387  
     etiology of, 387  
     occuli, 386  
     prognosis of, 388  
     symptoms of, 387  
     treatment of, 388  
 Spinal accessory nerve, anatomical considerations of, 129  
     paralysis of, 151  
     spasm of, 149  
 Arteries, inference, 342  
     posterior, 345  
 Cord, anatomical considerations of, 340  
     circulation in, 342  
     combined sclerosis of, 444. See also *Cerebral sclerosis*.  
     disabling transverse lesions of, table of symptoms, 323-326  
     glioma of, 385  
     hemorrhage into, 392  
     in brain tumors, 291  
     multifocal lesions of, 392  
     lesions of, combined, in anterior and posterior, 468  
     cross, table of symptoms in, 353-359  
     in tabes dorsalis, 431  
     localization of, vertical, 347  
     lesions of anesthesia in, 35  
     motor symptoms of, 347  
     of gray matter of, 388  
     of one lateral half of, 36  
     reflexes in, 349  
     sensory symptoms of, 349  
     trophic conditions in, 350  
     vascular disturbances in, 350  
     vertical localization of, 347  
     visceral symptoms in, 350  
     white matter of, 428  
     localization of, 340  
     relation of lesions and diseases of, 372  
     to body posture, 341  
     to vertebra, 341  
 Suggests, functions of, 348  
     relation of anesthesia to, 55  
     in entorhinal area, 56, 59  
 Subdural cysts of, 384  
 Syphilis of, 498. See also *Syphilis, spinal*.



- Spinal cord, thrombotic softening of, 364  
 transverse section of, 343  
 systematic source of substance of, 362  
 tumors of, 382  
 trunks, 784  
 fluid in cerebrospinal syphilis, 997  
 leptomeningitis, acute, 278  
 chronic, 281  
 meningeal hemorrhage, 282  
 meningos, tumors of, 382  
 meningitis, 276  
 nerves, division of, 280  
   histological changes in, 285  
   muscular symptoms of, 296  
   sympptoms of, 285  
   treatment of, 287  
 injuries and diseases of, 284  
 lesions of, in tabes dorsalis, 432  
 myelomeningitis, 276  
 special lesions of, 285  
 stretching in family ataxia, 476  
   in tabes dorsalis, 461  
 symptoms, in tabular leptomen-  
 ingitis, 95  
 syphilis, 495. *See also Syphilis,*  
*spinal.*  
 tumors, 382  
   course of, 385  
   diagnosis of, 385  
   location of, 386  
   morphological anatomy of, 383  
   progress of, 385  
   reflexes in, 384  
   symptoms of, 384  
   treatment of, 386  
 practice, 77  
   in cerebrospinal syphilis, 997  
   in diagnosis of cerebral hemorrhage,  
   279  
   in Landry's paralysis, 380  
   in leptomeningitis, 88  
 Spine, concussion of, 699  
 Spasmodic irritation, 525  
 Spasms, unlike chorea, 38  
 Spasmodic palsy, 173  
 Squint, importance of, 65  
 St. Vitus' dance, 566  
 Stahl case, No. 1, 716, 716  
   No. 2, 716  
 Stammering, 68  
 Staphylococcus aureus in chorea, 570  
   progress in chorea, 570  
 Status epilepticus, 61  
 Status epilepticus, 654  
 Stapes-shaped skull, 760  
 "Stepping," 320  
 Stenocardia, 51  
 Stenocardia, cerebral localization, 170  
 Stenocardic arrest, 51  
   treatment of, in tabes dorsalis, 441  
 Stereotyped movements, 752, 758  
 Stereotype in dementia senilis, 814  
 Strabismus, low in cerebellar disease, 180  
 Stigmata hereditaria, 698  
   of degeneracy in nervous disease, 21  
   of degeneracy in insanity, 699  
 Stimulants in leptomeningitis, 87  
 Strass venter, 192  
 Strain, physical and mental in etiology  
   of insanity, 725  
 Stream of thought, actions induced by  
   disorders of, 568  
 Streptococcus in chorea, 570  
   in leptomeningitis, 79  
 "Stroke," apoplectic, 216  
 Streptothrix in ophthalmic ptery, 557  
 Struma ophthalmica, 528  
 Strycin in anterior poliomyelitis, 367  
   in brain tumor, 270  
   in bronchial asthma, 144  
   in cerebral softening, 239  
   in chorea, 577  
   in ophthalmic ptery, 557  
   in nervous diseases, 152  
   in neuritis, 673  
   in neuroticosis, 698  
   in neuritis, 291  
   in polioencephalitis superior chronica,  
   152  
   in spinal progressive muscular atrophy,  
   412  
   in Thomsen's disease, 589, 794  
 Stupor, 762  
   motor, 760  
 Stuttering, 68  
 Subdural cysts of spinal cord, 354  
   space, 72  
 Subjective acoustic hearing of, 66  
 Suicidal tendencies, management of, 789  
 Sulphonal in chorea, 577  
   in insanity, 787  
   in mania, 798  
 Summary recommendations, 754  
 Supra-orbital ptery, 55  
 Suprasylvian nerve, lesions of, 287  
 Syllable-stuttering, 68  
 Syncope, 719  
 Symmetrical ataxia, 557  
 Sympathetic nerve, effect of division of,  
 110  
 Sympson, extension, 192  
   group of Weber, 112  
   inversion, 192  
   Henderson, 20  
   upward, 32, 193  
 Symptomatic disorders, 965  
 Symptoms of a disease, 196  
   diffuse, 194  
   localized, 194  
 Synclitically, 719  
 Syndrome, Benedikt-Weber, 188  
   paired, 557  
   thalamic, 587  
 Synergy, 29  
 Syphilis, cerebral, 492  
   arterial form of, 496  
   diagnosis of, 495  
   general symptoms of, 493  
   granulations form of, 790  
   hereditary is, 494  
   meningeal form of, 495  
   meningeal symptoms of, 496  
   prevalence of, 490

- Syphilis, cerebral, prognosis of, 496**  
 salivarian in, 502  
 special symptoms of, 495  
 spinal fluid in, 497  
 treatment of, 498, 501  
 Wassermann reaction in, 497  
**hereditary cerebrospinal, 501**  
 in etiology of ulcers, 577  
 of insanity, 725  
 of pseudotuberculous, 821, 831, 832  
 inherited, predisposing to nervous disease, 18  
 of cranial nerves, 493  
 of nervous system, 490  
   acquired, 491  
 spinal, diagnosis of, 500  
   prognosis of, 501  
   treatment of, 503  
**Syphilitic stasis paraplegia, 500**  
 cerebral arteritis, 492  
   meningitis, 492  
   cerebritis, 492  
   meningocerebritis, 499  
 mental disease, 496  
 myelitis, 500  
 neuritis, 501  
 pseudoparesis, 497  
 softening of cord, 500  
 spinal meningitis, 499  
   paralysis, 500  
 tumors of brain, 259  
**Syphilophobia, 497**  
**Symphalangia, 414**  
**Syringomyelia, 399**  
   anesthesia in, 402  
   arthropathies in, 404  
   atrophy in, 403  
   clinical forms of, 405  
   course of, 404  
   diagnosis of, 406  
   etiology of, 399  
   mental anatomy of, 400  
   Mott's type of, 405  
   motor disturbances in, 403  
   prognosis of, 400  
   sensory disturbances in, 402  
   symptoms of, 401  
   treatment of, 406  
   trophic features of, 403  
   truncal symptoms of, 401  
   vegetative symptoms of, 404  
**Syriacusville dissimulation, 402**

**Taxes, continued, 464**

- desails, 429**  
 anisotropia in, 454  
 analgesia in, 440  
 aneurysm in, 491  
 aspin in, 453  
 ataxia in, 450  
 auditory symptoms in, 445  
 blood in, 450  
 bones in, 450  
 cerebral disturbances in, 445  
 constipation in, 447  
 course of, 450

- Tuber dorsalis, cramps in, 440**  
 crises in, 446  
 diagnosis of, 455  
 diarrhea in, 447  
 differential diagnosis of, 458  
 disorders of generative function in, 448  
   of intestines in, 447  
   of nutrition in, 450  
   of osseous system in, 450  
   of respiratory apparatus in, 449  
   of skin in, 454  
   of stomach in, 446  
   of urinary apparatus in, 447  
   of vascular apparatus in, 449  
 disturbance of the reflexes in, 442  
 etiology of, 429  
 fat-foot in, 453  
 gastric crises in, 446, 474  
 general paresis and, 826  
 girdle sensation in, 449  
 glycosuria in, 448  
 herpes zoster in, 454  
 hyperalgesia in, 441  
 impairment of stereognostic sense in, 441  
 impotence in, 448  
 involuntary movements in, 448  
 juvenile, 458  
 labyrinthine disturbance in, 446  
 hysterical crises in, 449  
 lightning pains in, 429  
 mercury in, 460  
 mental anatomy of, 431  
 motor disturbances in, 426  
 muscles in, lack of tonicity of, 443  
 muscular atrophies in, 454  
 apoplexy in, 458  
 nasal crises in, 446  
 neuroleptism in, 460  
 nephritic crises in, 448  
 optic nerve in, 445  
 pains in, 429  
 palsies in, 458  
 perforating ulcer in, 454  
 pharyngeal crises in, 449  
 phenacetin in, 463  
 prognosis of, 429  
 ptosis in, 449  
 pupils in, 444  
 rarifying osteitis in, 451  
 rectal crises in, 447  
 resection of nerves in, 443  
 Robertson's sign in, 445  
 Romberg's sign in, 426  
 salivarian in, 490  
 sensory disturbances in, 429  
 spontaneous fractures in, 450  
 spirit in, 441  
 stroke in, 449  
 symptoms of, 426  
 tabetic arthropathy in, 451  
 tubulation of, 455  
 temperature in, 450  
 treatment of, 460  
 trophic cutaneous disorders in, 454  
   disorders in, 450



- Tubes dorsalis, atropia in, 463  
 valvular disease in, 449  
 varieties of, 456  
 visceral disorders in, 446  
 visual disturbances in, 444  
 vomiting in, 446  
 Westphal's sign in, 442  
 spasmodic, 464
- Tubercle arthropathy, 451  
 crura, 446  
 crura, 441  
 fractures, 450  
 joint, 452
- Ticks, oticulae in leptomeningitis, 80  
 in tubercular leptomeningitis, 94
- Tachycardia, 445
- Tactile sense, 58  
 testing of, 59
- Taste, center for, 171  
 electrical testing of, 69  
 examination of, 68  
 inflammation of, 128  
 lesions of, 743  
 in facial paralysis, 126  
 loss of, 67  
 perversion of, 67  
 subjective sensations of, 67
- Teeth, anomalies of, 713
- Temperature changes in insanity, 704  
 examination of, 25  
 in anterior poliomyelitis, 203  
 in cerebral hemorrhage, 211  
 in leptomeningitis, 83  
 in spinal leptomeningitis, 280  
 in tubercular leptomeningitis, 92  
 localized elevation of, as a symptom in brain disease, 195
- Tender points in sciatic neuritis, 311  
 of Valleix, 670
- Tenderness as a symptom, 58  
 of brain disease, 195  
 in leptomeningitis, 83  
 in multiple neuritis, 324  
 in sciatic neuritis, 311
- Tenckin reflex in leptomeningitis, 84  
 in tubercular leptomeningitis, 92
- Tenckin, trochic disorders of, 46
- Tetanus of brain, 269
- Test, Bielsky's labyrinthine, 136  
 Ewald's labyrinthine, 136  
 epithalamic, in tubercular meningitis, 95  
 thermic, for labyrinthine disease, 136  
 tubercular, in tubercular meningitis, 95
- Tetania, 542. See also Tetany.
- Tetanus, 561  
 antitoxin in, 563  
 cerebral and in, 563  
 cutaneous, 561  
 cutaneous, 561  
 diagnosis of, 562  
 etiology of, 561  
 etiology in, 563  
 head, 562  
 hydrophobia, 562  
 Kerner's sign in, 561  
 magnesium sulphate in, 563
- Tetanus, morbid anatomy of, 561  
 asphyxia in, 563  
 neonatorum, 562  
 progress of, 562  
 paralytic, 562  
 symptoms of, 561  
 treatment of, 563  
 varieties of, 562
- Tetany, 542  
 course of, 546  
 diagnosis of, 546  
 etiology of, 542  
 leg sign in, 545  
 progress of, 544  
 symptoms of, 544  
 treatment of, 546
- Thalamic syndrome, 187
- Thermic sense, testing of, 51  
 tests for labyrinthine disease, 136
- Thermosensation, 54
- Thermo-anesthesia, 54
- Third nerve, affection of, in leptomeningitis, 83  
 anatomical considerations of, 408  
 effect of division of, 109
- Thomson's disease, 588  
 diagnosis of, 591  
 etiology of, 588  
 morbid anatomy of, 588  
 symptoms of, 588  
 treatment of, 591
- Thomson's nerve, posterior, lesions of, 286
- Thought inhibition, 752
- Thrombosis a cause of cerebral softening, 222, 226  
 of arteries of spinal cord, 364  
 of veins, 232. See also *Sinus thrombosis*.
- Thrombotic softening of spinal cord, 364
- Thyroid extract in myxedematous stony, 945  
 feeding in hydrocephalus, 375  
 glial, trophoblastic related to, 518  
 in idiopathic diabetes, 517  
 treatment, 526
- Thyroiditis, 528
- Tie, motor, 434  
 shockerum, 31, 616  
 etiology of, 615  
 treatment of, 616  
 varieties of, 616
- Tinnitus, 131, 632
- Tie-sign of Blyskowski, 37  
 in myelitis, 369, 370
- Tongue, anomalies of, 713  
 motor center for, 169  
 paralysis of, 153
- Tonic contractions, 32  
 extreme in cerebral disease, 190  
 spasm, 31
- Typical symptoms in brain disease, 195
- Typhoid, mental, 159, 626  
 spasmodic, 149, 456
- Tons palatine, 709, 711
- Toxic blindness, 187  
 influence in etiology of insanity, 713
- Trance, hysterical, 635

- Transitory mania, 796  
 Transverse myelitis, 300  
 Traumatic leukoencephalitis, 728  
   idiocy, 873  
   insanity, 728  
   neuritis, 292  
   neurones, 566  
   psychosis, 662  
 Triceps, 36  
   family, 354  
   fibillary, 30  
   handwriting in, 31  
   importance of, 30  
   intention, 30  
   testing for, 31  
   volitional, 30  
 Trichiasis in cerebral hemorrhage, 221  
 Trichomoniasis, 88  
 Triceps reflex, 35  
 Trifacial nerve, anatomical considerations of, 116  
   cortical diseases of, 118  
   disease of branches of, 119  
   diseases of, 116  
   typical disease of, 118  
   peripheral (extracranial) affections of, 118  
   tetralgia, 871  
   neuritis, 120  
 Trigeminal. *See* Trifacial nerve.  
 Tripsocephalus, 704  
 Trisyllabic isogenic perioris, 148  
   in Raynaud's disease, 349  
 Trochlear chiasm, 577  
   in epilepsy, 666  
   in insanity, 282  
   in mania, 798  
   in neurosthenia, 669  
 Trochlear, 361  
 Trochlearic point, 311  
 Trophic conditions, 39  
   diseases in insanity, 592  
 Trophoedema, chronic boundary, 569  
 Trophic nervous related to hypophysis  
   cervici, 596  
   to parathyroid gland, 518  
   to thyroid gland, 518  
 Trovimen's sign of tetany, 545  
 Trunk, center for movements of, 169  
   nerves of, lesions of, 306  
 Tubercle of the brain, 256  
 Tubercular meningitis, 90. *See also*  
   *Leptomeningitis, tubercular.*  
 Tuberculin in paralytic dementia, 835  
   test in tubercular meningitis, 95  
 Tuberculosis in etiology of insanity, 726  
 Tumors in cerebellopontine angle, cystic  
   tumors presented by, 191  
   in frontal region, vertigo with, 190  
   of brain, 264  
   changes in cord in, 261  
   course of, 267  
   diagnosis of, 264  
   etiology of, 256  
   hematoma in, 262  
   hemiplegia in, 265  
   pathological anatomy of, 256  
 Tumors of basis, prognosis of, 269  
   symptoms of, 264  
   cystic, 259  
   treatment of, 269  
   vertigo in, 264  
   visual field in, 64  
   vomiting in, 264  
   of nerves, 261. *See also* *Neuromat.*  
   of nasal gland, 518  
   of spinal cord, 382  
   meninges, 382  
   pituitary, with acromegaly, 266  
   with hydrocephalus, 251  
   pseudo-cerebral, 267  
 Tuning-fork in testing hearing, 65  
 Turkish bath in multiple neuritis, 335  
 Twelfth cranial nerve. *See* *Hypoglossal*  
   *nerve.*  
 Typical bacillus in leptomenigitis, 79  
 Ulnar nerve, lesions of, 309  
 Uremic fits, 651  
 Unproductive mania, 791  
 Unsymmetrical hypertrophies, 558  
 Urine, condition of, 26  
   in insanity, 765  
   in leptomenigitis, 84  
   incontinence of, 25  
 Ursemia in *tubercularis*, 463  
 Uremia, giant, 552  
   in leptomenigitis, 84  
 Valves, anatomical considerations of, 138  
   carotid branches of, 145  
   disease of, 138  
   gastric branches of, 148  
   laryngeal branches of, 159  
   pharyngeal branches of, 159  
   pulmonary branches of, 141  
 Valvula in urethralia, 581  
 Valleys, sensitive points of, 626  
 Vascular disorders in insanity, 765  
   neurones, 547  
 Veins, cerebral, anatomy of, 231  
 Venereal history, importance of, in diag-  
   nosis of nervous disease, 19  
 Venosclerosis in cerebral hemorrhage, 220  
 Verbal operations for idiocy, 916  
 Verberation, 753, 794  
 Verid in insanity, 787  
   in mania, 677  
 Verücktheit, 687  
 Vertical segments, relation of medial  
   points of gaze to, 59  
 Vertical localization of a cord lesion, 347  
   point, 172  
 Vertigo as symptom, 194  
   aural, 194. *See also* *Aural vertigo.*  
   in brain lesions, 194  
   in cerebellar disease, 194  
   in disease of labyrinth, 66  
   in epilepsy, 652, 654  
   in hysteria, 685  
   in multiple cerebello-spinal sclerosis, 487  
   in tumors of brain, 264  
   of frontal region, 190



- Vestibular nerves, diseases of, 134
- Violence in insane, management of, 780
- Vulva-reflex, 38, 448
- Visceral diseases, areas of pain and, 36-38, 72
- Vision, center for, 178  
 electrical testing of, 49  
 diseases of, 742  
 in multiple neuritis, 324  
 testing of, 61
- Vital spleen, 180  
 felt, 64  
 testing of, 64  
 functions, cortical representation, 170  
 in brain tumor, 265  
 in nervous diseases, 61  
 tract, 59
- Volitional impulse, 30
- Yawning as a cerebral symptom, 194  
 in leptomeningitis, 81  
 in lesions of spinal cord, 354  
 in tabes dorsalis, 146  
 in tubercular meningitis, 92  
 in tumor of brain, 264  
 projectile, as a symptom, 180
- Von Pirquet's test in tubercular meningitis, 95
- WADSWORTH, 687
- Wassermann reaction, 25  
 in cerebral syphilis, 497  
 in paralytic dementia, 831
- Wasting palsy, 908
- Weakness, paralytic, in leptomeningitis, 82  
 of polio, 752
- Weber, symptom group of, 113
- Weber's test, 448
- Weissbach's diplococcus in leptomeningitis, 79
- Wernicke-Hoffmann type of muscular atrophy, 424
- Wernicke's homonymous pupillary reaction, 191
- Wernicke's sign, 34
- Westphal's sign, 442
- Whisky in angina pectoris, 148
- White cerebral softening, 223  
 section of cord, lesions of, 428
- Willebrand's lateral ear, 716  
 ear, 716, 718
- Wilson's disease, 187, 383
- Wolf families, 174  
 stability of, 178
- Wundt-Jacobson method of testing intelligence and emotions, 772
- Word-blindness, explanation of production of, 302  
 lesions causing, 171
- Wrist syncope, 174
- Wrist-fulness, 152, 178  
 lesions causing, 171
- Wurmian bones, significance of, 700
- Wrist, center for movements of, 169
- Wrist-clonus, 55
- Writer's cramp, 288, 293  
 course of, 297  
 diagnosis of, 298  
 etiology of, 293  
 motor disorders of, 260  
 pathology of, 294  
 prognosis of, 298  
 sensory disorders in, 297  
 symptoms of, 290  
 treatment of, 298
- Written speech, paraphasia of, 91
- Wryneck, spasmodic, 149
- X-ray in cerebral rube in meningitis of basal ganglia, 306
- YACOWS cerebral-softening, 223
- Zeeb, 756. See also *Herpes zoster*.
- Zoster, 336. See also *Herpes zoster*.













[illegible]

Demco 293-5



RC341  
921 C



